A
PRACTICAL HANDBOOK
ON THE
MUSCULAR ANOMALIES OF THE EYE

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PREFACE.

THE aim of the authors in writing this book has been to present to beginners in ophthalmic work, the principal facts in the diagnosis and treatment of abnormal states of the eye-muscles.

The subject-matter is an elaboration of a short series of lectures, delivered in successive winter courses at the Philadelphia Polyclinic. Stimulated by the interest displayed by the classes, and their constant inquiries for a practical text-book on the subject, the authors have endeavored to give book-form to their ideas about the muscular anomalies of the eye. They have sought to clothe their thoughts in language easily understood; to avoid discussions and speculations; to emphasize methods that have stood the test of their own experience; and, without giving the volume the proportions of a book of reference, to omit no important data that have been recognized as trustworthy.

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PART I.

INTRODUCTORY.

CHAPTER I.

ANATOMY.

THE study of the anatomy and physiology of the external muscles of the eyeball, in the human subject, embraces a consideration of their structure, their innervation, their relations to the orbit and to the ball, and their dynamics.

The external muscles as a whole, with the exception of the inferior oblique, form an irregular cone, the angle of which is at the apex of the orbit while its base embraces the ball near the anterior plane of the orbit. If the orbital axes were continued backward they would cross at an angle of 20 37 degrees,[1] the variation depending upon race, age, and individual characteristics. The direction of the muscular cone depends upon the degree of divergence of the orbits, their depths, and perhaps the shape of the ball. The total length of the muscles, the inferior oblique excepted, is affected by the increase in depth of the orbit, and particularly by the increase in the width of the anterior segment of the orbit that occurs during the development of the eye, thus determining the size of the "contact arc" or that segment of the circumference of the eyeball that is in actual contact with the muscles or their tendons. As the divergence of the orbits increases, the contact arc of the external rectus undergoes a considerable increase and that of the internal rectus a considerable decrease.

Weiss,[2] in speaking of the growth of the eye from infancy to maturity as compared with the brain's growth during a corresponding period, says: "As to the extrinsic muscles, the relation is much the same in the new-born as in the adult, the chief difference being in the distance of their insertion from the cornea. For instance, the distance of insertion of the rectus internus in a 20-year-old has increased in a proportion of 1 : 1.63, while the increase in distance from the cornea of the other recti muscles during the same period shows a proportion of but 1 : 1.37. The increase in distance of the muscular insertions from the entrance of the optic nerve bears out a similar proportion, from which it follows that the growth of the sclera in its anterior and posterior halves from infancy to adult age is about equal."

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[1] L. Weiss, *Arch. of Ophthal.*, July, 1896.

[2] *Bericht über die 24. Versamml. der Ophthal. Gesell., Heidelberg.*

The orbital divergence, above referred to, creates a more or less closely corresponding divergence of the ocular axes which has an important mechanical relation to the position of rest of the eyes, presumably assumed during sleep or other narcosis, and to the action of the muscles in associated movements. It forms the basis for the opinion of those who argue that the position of rest is divergence, and that parallelism and convergence are maintained only at the expense of muscular contraction. The center of rotation of the eyeball lies in the axis of vision at a point averaging 13.45 mm. behind the summit of the cornea.

THE MUSCLES INDIVIDUALLY.

The eye is moved by 6 muscular bands symmetrically placed, 4 straight and 2 oblique.

The *external rectus*, supplied by the 6th nerve, arises from two heads—the larger from the greater wing of the sphenoid in the external and lower border of the sphenoidal fissure; the smaller from the outer margin of the optic foramen. These two heads soon become united to form the body of the muscle which runs forward and outward, almost in contact with the outer orbital wall for nearly half its length, and loosely bound to it by connective tissue. It is attached anteriorly to the sclera by a tendon 3.7 mm. in length, 9.2 mm. in width, in a vertical line 7 mm. from the outer limbus. Its area in the thickest portion of the muscle equals 16.73 sq. mm. Its length is 40.6 mm. (averages for adult). The *external* rectus controls the outer half of the field of fixation and rotates the cornea horizontally temporalwards. It has no action in tilting the upper end of the vertical meridian of the cornea either inwards or outwards.

The *internal rectus*, supplied by the 3d nerve, arises in a tendon common to it and to the inferior rectus from the inner margin of the optic foramen, runs forward, lying close to the inner wall of the orbit and is inserted into the sclera by a tendon 8.8 mm. in length and 10.3 mm. in width, at a distance of 6.5 mm. from the inner limbus. The area of its thickest portion is 17.39 sq. mm., and its length is 40.8 mm. It controls the inner half of the field of fixation, rotates the cornea horizontally nasalwards, and has no torsional action.

The *superior rectus*, supplied by the 3d nerve, arises by a tendon common to it and to the inferior and internal rectus at the upper margin of the optic foramen. In its course forward it perforates the orbital fat lying just under the levator palpebræ, and, after making a diverticulum of Tenon's capsule on its upper meridianal surface, is inserted into the sclera anterior to the equator and 8 mm. posterior to the corneoscleral border, by a tendon 5.8 mm. in length and 10.6 mm. in width. Its largest area is $11\frac{1}{3}$ sq. mm. and its length 41.8 mm. From these figures it will be seen that the superior rectus is the weakest of the straight muscles. It controls the upper half of the field of fixation, turning the eye vertically up, this vertical movement increasing as the eye is abducted and diminishing as the eye is adducted; it also rotates the cornea horizontally nasalwards and tilts the upper end of the cornea in, more in adduction, less in abduction.

The *inferior rectus*, supplied by the 3d nerve, has the same origin as the internal. It lies close to the floor of the orbit and is attached to the sclera by a tendon 5.5 mm. long and 9 8 mm. wide, at a distance of 7.2 mm. from the limbus. Its largest area in section is 15.85 sq. mm. and its length is 40 mm. It controls the lower half of the field of fixation, turning the eye vertically down, the vertical movement increasing with adduction, diminishing with abduction. It also turns the eye laterally in and rotates the upper end of the vertical meridian of the cornea out, these effects increasing as the eye is adducted, decreasing as it is abducted.

The *superior oblique*, a long fusiform muscle supplied by the 4th nerve, has its origin a little above the common origin of the recti from the less wing of the sphenoid, passing forward and upward to the inner angle of the orbit, where as a tendon it plays in, and is supported by, a fibro-cartilaginous ring in the fossa trochlearis. Upon resuming its course (the direction of which is now backwards, downwards and outwards at an angle of 53°), it passes beneath the superior rectus, to be attached to the sclera midway between the cornea and the optic nerve foramen 16 mm. from the limbus. The insertion forms nearly an antero-posterior line, the large part of the tendon being attached posterior to the vertical equator of the globe. The length of the tendon, which commences before the ring is reached and is continued to the scleral end, is 19.5 mm., and the width of the insertion varies from 6.8 to 14 mm. It moves the eye down and out and rotates the upper end of the vertical meridian inward, both movements increasing in abduction, diminishing in adduction.

The *inferior oblique*, supplied by the 3d nerve, arises from the anterior portion of the middle of the floor of the orbit in a depression in the superior maxillary bone just external to the anterior end of the lacrimal groove. As a thin, narrow muscle, it passes outwards, backwards and upwards, beneath the inferior rectus, and is inserted by a horizontal attachment, about 10 mm. in width, into the sclera between the inferior and external recti on the posterior hemisphere of the ball, 17.3 mm. from the limbus. It controls the upper outer half of the field of fixation, turning the eye (vertically) upwards, which effect increases in adduction, and diminishes in abduction;[3] it also turns the eye (laterally) outwards, more in abduction, less in adduction.

The *levator palpebræ*, while not strictly one of the extrinsic muscles of the ball, is closely allied, anatomically and pathologically, to them and should be included in a description of the muscles of the orbit. It arises from the under surface of the less wing of the sphenoid above the optic foramen, passes forward above and in close juxtaposition to the superior rectus in the first half of its course, and is inserted into the upper edge of the tarsal cartilage by an aponeurosis as broad as the cartilage. These two muscles are so intimately associated in their location and course that contraction of one involves partial contraction, at least, of the other. A similar association is noticed

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[3] Volkman's results of measurement of movements, quoted by Weiland, *Archives of Ophthal.*, January, 1898, are:

Rect. Ext.	moves	eyeball	out, up;	rotates	upper	cornea	inward.
Rect. Int.	"	"	in, up;	"	"	"	outward.
Rect. Sup.	"	"	up, in;	"	"	"	inward.
Rect. Inf.	"	"	down, in;	"	"	"	outward.
Obl. Sup.	"	"	down, out;	"	"	"	inward.
Obl. Inf.	"	"	up, in;	"	"	"	outward.

between the inferior oblique and inferior rectus, due to connecting bands of delicate fibrous tissue. (Kœnigstein.)

The ball is enveloped in the major part of its extent by two layers of fibrous tissue, united at the anterior and posterior extremities to form a closed sac, known as Tenon's capsule. Commencing 5 cm. from the optic foramen, the membrane passes forward, loosely attached to the sclera, and anteriorly to the conjunctiva by connective tissue, and ends a few millimeters posteriorly to the corneal border. The opposing surfaces are lined by endothelium and form a lymph space. Posteriorly the ciliary nerves and tendons of the oblique muscles, and anteriorly the tendons of the straight muscles, perforate the capsule, without, however, destroying the continuity of its walls, or its function as a lymph space. Minute channels pass from Tenon's capsule through the sclera to communicate with the suprachoroidal lymph space and indirectly with the perineural lymph spaces. It thus forms an important link in the chain of lymph spaces connecting the eye in all its parts with the intracranial lymph system. The tendons of the muscles and the trunks of the ciliary nerves are covered for a short distance from the globe by diverticula of Tenon's capsule, which, in the case of the former, strengthen their scleral attachments.

In operations on the muscles, the prolongations of the capsule forming enveloping sheaths must not be disregarded. Difficulty in picking up the tendons with the strabismus hook is often encountered, because the incision has failed to divide both layers of the capsule, and the effect desired of a tenotomy is not secured, because attention has not been given to the radical supporting attachments derived from the capsule and the check ligaments which are a portion of the orbital fascias. Serious inflammations (tenonitis), with extension of the inflammatory process backward, may result from traumatism to this serous envelope.

The space between the ball and the orbital walls, not occupied by muscles and nerves, is filled with fat in great part, and by connective tissue. The connective tissue of the orbit is so arranged as to form light fibrous bands, probably containing elastic tissue, supporting and steadying the muscles during action.

VASCULAR SUPPLY.

The *vascular supply* to the muscles is derived from the muscular branches of the ophthalmic artery, one of the terminal branches of the internal carotid.

The ophthalmic artery at its origin is 2 mm. in diameter. It passes through the optic foramen under the optic nerve, supplying its sheaths and perineurium, later perforating and supplying the nerve-substance itself. Soon after its entrance into the orbit, it gives off the muscular branches. All the blood-vessels of the orbit are characterized by their tortuous course and their lax attachments to flexible structures. They are thus enabled to accompany the movements of the ball without tearing.

The *muscular arteries* are two in number; the smaller passing up and medianwards, the larger downwards and laterally, supplying the muscles in their course. They give off the anterior ciliary branches which perforate the tendons of the muscles and the sclera, and supply the ciliary region with blood. (Section of one of these arteries in tenotomy often gives rise to profuse but never alarming hemorrhage.)

The arteries are accompanied by veins situated similarly and described under similar names.

CHAPTER II.

PHYSIOLOGY.

INNERVATION.

THAT the different movements of the eye are represented in the cerebral cortex, was suspected and experimentally shown with some degree of probability by Majendie early in the century. Since his time a few observers, notably Ferrier, Munk, Marchi, Mercier, and Warner, have endeavored to solve the physiology of this question. Perhaps no worker has shed more light on the subject than Risien Russell, whose experiments on dogs three years ago

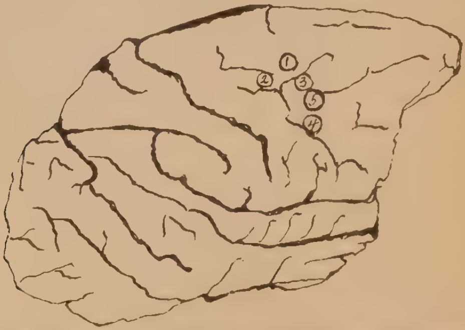

FIG. 1.—The external surface of the right half of the brain of the ape (Macacus Sinicus). (After Russell, *Journal of Physiology*, vol. 17, p. 8.)

1. Upward movement of both eyes.
2. Downward movement of both eyes.
3. Movement of both eyes upward and to the opposite side.
4. Movement of both eyes downward and to the opposite side.
5. Convergence.

The numbers denote the chief foci, excitation of which evoked movements c the eyes other than conjugate turning to the opposite side from the hemispher stimulated.

led him to conclude that the cerebellar cortex played no little part in ocular movements, and that it was associated with the cerebrum in these functions. The accompanying drawing, Fig. 1, based on Russell's experiments, indicates the areas which are supposed to preside over the different eye-movements. It will be observed that they are just anterior to the large motor area, and, moreover, that they are close to the facial area, and, on the left side, in intimate association with the speech-center of Broca. The ocular effect produced by ablation of the different lobes of the cerebellum is paralytic to different associated eye-movements, but whether this arrest of eye-movements so produced proceeds from direct influence of the cerebellum on the eye-muscles or arises indirectly through the loss of the cerebellar influence over the cerebrum, is uncertain.

Nystagmus following ablation of cerebellar areas is, according to Russell (*loc. cit.*), either irritative or paralytic, probably the former, through the weakness of the muscles related to the destroyed area. Two antagonistic influences appear to be exerted on the extrinsic muscles; *e. g.*, the left lateral lobe of the cerebellum is supposed to antagonize the influence of the eye-area of the left cerebral hemisphere over the external muscles of the right eye, and vice versa. On the other hand, the right lateral lobe of the cerebellum and the eye-area of the left cerebral hemisphere are said to act conjointly in inducing movements of the eyes in one direction, and conversely, the left lateral cerebellar lobe and the eye-area of the right cerebral hemisphere are similarly associated in their functions. If, as has been shown, every movement of the eyes, individually or conjointly, demands the combined action of at least two, if not more, of the ocular muscles, it would appear that every excursion performed by the eyes is in greater or less degree an associated movement, and if this be admitted, the importance of the foregoing experiments cannot be over-estimated.

The cortical areas, above referred to, are the highest centers for movements of the eye. There is another region intimately associated with ocular movements, about which we are in possession of much more knowledge, namely: the region of the 4th ventricle, one of the subcortical areas.

In general terms, the cell-groups constituting the nuclei of the nerves governing the ocular muscles lie in the gray matter of the floor of the aqueduct of Sylvius and of the 4th ventricle, the major gathering being beneath the corpora quadrigemina.

The 3d, 4th, and 6th cranial nerves, known also as the oculo-motor, pathetic, and abducens nerves, together supply the external ocular muscles. The 3d nerve also gives off a branch to the ophthalmic or lenticular ganglion, forming its motor root to the interior ocular muscles. The structure of the 3d nerve is so composite that volumes of controversy have been expended in attempting to accurately locate and arrange its individual nucleoli. The scheme or hypothesis which explains the largest number of 3d nerve nuclear problems is, probably, that of Stuelp,[1] Fig. 2.[2] (Page 19.)

It differs slightly from Starr's and that of Kahler and Pick. Stuelp's scheme agrees with all others in placing the nucleoli related to the iris and ciliary muscle (the intra-ocular muscles) in the most anterior part of the 3d nerve nucleus. Then follow from before backwards the nucleoli governing the action of the four external ocular muscles supplied by the 3d nerve. Directly back of the last of these lies the nucleus for the trochlearis or 4th nerve (superior oblique), and close upon this comes the aggregation of cells representing the 6th nerve nucleus (external rectus). The nuclei lie along the middle of the floor of the 4th ventricle, and although decussation of the root fibers has not been positively demonstrated, Bruce[5] believes this to be the case whereby the different nuclei are brought into relation with each other. They all bear certain relations to the cortical areas above mentioned.

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[1] *Archiv f. Ophthalmologie*, July, 1895.

[2] Some authorities prefer Perlia's scheme.

As the fibers of the 3d nerve leave the mass of gray substance which lies along each side of the median line, in the anterior part of the 4th ventricle, they converge as they pass downward through the red nucleus, the sensory and the motor tracts to pierce the dura mater below the posterior clinoid process; the nerve-trunk there passes along the outer wall of the cavernous sinus, and divides into two branches. The superior division supplies the superior rectus and levator palpebræ, the inferior division separates into three branches, one going to the internal

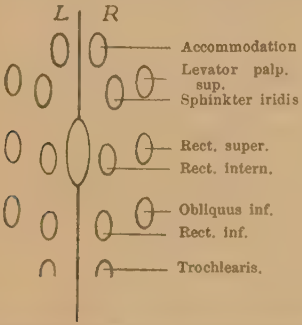

Fig. 2. Stuelp's scheme of the arrangement of the 3d and 4th nerve nucleoli.

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[5] Illustrations of the Nerve Tracts of the Mid and Hind Brain.

rectus; and the third, and largest, to the inferior oblique. The nerve-trunk also receives filaments from the cavernous plexus of the sympathetic, and its superior division is not infrequently connected with the ganglionic branch of the nasal nerve.

The 4th, pathetic, or trochlear nerve, supplies the superior oblique muscle. Its deep origin can be traced to the nucleus which lies beneath the aqueduct of Sylvius, behind and on a lower level than the 3d nerve nucleus. The fibers pass gradually to the dorsal aspect of the aqueduct and at the valve of Vieussens decussate with the fibers of the opposite side. Its connections with the nucleus of the 6th nerve, which is described by some authors, is doubted by Bruce (*loc. cit.*), who regards it as more probable that fibers may go to that division of the opposite oculo-motor nucleus which supplies the inferior rectus. The nerve-trunk pierces the dura mater near the posterior clinoid process, passes with the 3d nerve along the outer wall of the cavernous sinus and enters the orbit through the sphenoidal fissure, being the highest of the nerves passing through the orbit. It also receives filaments from the cavernous plexus of the sympathetic, and transmits a twig to the lacrimal.

The nucleus of the 6th or abducens nerve consists of a column of multipolar ganglion cells lying immediately under the ependyma of the 4th ventricle on either side of the median sulcus. It is bordered on its inferior, inner and upper sides by the first, second, and third portions of the facial nerve. The fibers run obliquely downwards to emerge at the lower border of the pons. The connections of the 6th nucleus are said by Bruce (*loc. cit.*) to be as follows: with the second part of the root of the facial nerve and the segment of the 3d nerve nucleus supplying the internal rectus; with the auditory nucleus and with the cortex of the opposite cerebral hemisphere, and perhaps with that of the same side. The nerve penetrates the dura mater on the basilar surface of the sphenoid bone, passes through the posterior or clinoid processes, enters the cavernous sinus, and finally enters the orbit by way of the sphenoidal fissure. It also receives filaments from the carotid and cavernous plexus of the sympathetic nerve, from Meckel's ganglion, and from the ophthalmic nerve.

THEORY OF INNERVATION.

To outline the structure and function of each of the individual eye-muscles is a relatively simple matter; yet it is by no means so easy a task to give a satisfactory explanation of the innervational state of the eye-muscles corresponding to all the positions the eyes may assume in monocular or binocular fixation. The theory that has gained the widest acceptance is the so called innervational theory, so ably championed by Hansen Grut, twenty years ago. It provides that every contraction of a muscle or set of muscles to produce ocular movement in a given direction is accompanied by relaxation of the muscle or set of muscles that are directly antagonistic to the contracting muscles, which relaxation keeps even pace with the contraction going on in the muscles producing the deviation. As the eyes are capable of being easily rotated there must, in order that single vision be preserved, be a balance between the forces that tend to move the eyes in various directions. This balance is possible only under two conditions: (1) that all muscles be slack and exert no tension (an untenable hypothesis); or (2) that all the muscles so tend to contract as to cause an equal rotational strain in all directions. Hence, we suppose that the muscles are always in a state of partial tonic contraction, at any rate during the period of full consciousness.

There is no limitation to the directions in which the eye can be rotated by the action of one or more muscles, and under certain circumstances a contraction of all the muscles produces a slight enophthalmos.

The active muscles in the following movements from the primary position are: *in*, internal, superior, and inferior rectus; *out*, external rectus, superior and inferior oblique; *up*, superior rectus, inferior oblique; *down*, inferior rectus, superior oblique; *in up*, internus, superior rectus, inferior oblique; *up–out*, superior rectus, inferior oblique, external rectus; *down–out*, inferior rectus, superior oblique, external rectus; *down-in*, superior oblique, inferior rectus, internal rectus.

These are the eight principal secondary positions, and in all the degrees of the circle between them the rotation will be accomplished by the combined action of the muscles normally turning the cornea to those positions. The extent of the turnings in the field of fixation vary, according to different observers. The average is probably as follows:

up	up-out	up-in	out	in	down	down-out	down-in
40–50	35–50	40–55	35–55	40–48	40–70	35–60	25–60

(Duane.)

The discrepancies arise from variations in the normal power of the muscles of one individual as compared with another, and the amounts of attention and effort of which an individual is capable. Also in determining the rotation by Stevens's tropometer, the form of the ball must be taken into consideration. Such wide variations, as the averages given above, are confusing to one who endeavors to determine whether a certain movement is normal or otherwise.

Associated Movements. — During normal physiologic activity of the muscular apparatus (innervation), every movement of one eye is accompanied by a simultaneous and equal movement of the other. We cannot at will, for instance, turn one eye to the left and the other to the right, one eye up and the other down, but in every response to the will (conveyed through the channels of the basal centers), the nuclei and the trunks of the nerves on both sides demonstrate by their associated functions the conjoined action of the nuclei of two or more nerves. This joint action is the result of connections between the nuclei by means of fine fibers that have been physiologically studied and microscopically examined.

Convergence.—From parallelism the visual axes may be directed toward each other until they cross at an angle of from 43° to 60°. As has been stated before, parallelism of the visual axes is not the position of rest, but the position of equilibrium ; that there is some contraction of the abductors during convergence seems clear, because the eyes are held steady in the orbits, and because in full paralysis of the internus the affected eye is diverged. The muscles concerned in convergence are the same in each eye, viz., the internal, the superior, and the inferior recti, all of which are controlled by the 3d nerve, and the iris and ciliary muscle contract in equal measure. No separate action of the levator palpebræ or the inferior oblique can be recognized.

Abduction or Divergence.—The muscles concerned are the external rectus and the superior and inferior oblique, governed respectively by the 6th, 4th, and 3d nerves. The fact that abduction embraces the functions of several nuclei in the aqueduct of Sylvius proves conclusively the existence of internuclear connections. The eyes may be diverged from each other (under the influence of prisms, bases *in*) as much as 7° or 8°, and in this artificial position of greatest possible divergence, convergence may be said to begin; hence, the range of convergence reaches from the widest possible degree of divergence through parallelism up to the nearest point at which a very small object can be seen single (from ¾ to 2½ inches in front of the eye), "the fusion near point." In inverse manner, divergence begins at the fusion near point, extends outwards through parallelism to the greatest possible divergence of the visual axes (generally 7° to 8° of prism), and may hence be said to be identical with convergence both in range and in equivalence. Many students have an incorrect conception of the relation of convergence to divergence obtained from the published statements that adduction is three or four times stronger than abduction. This is because it is not made clear that these measurements are taken at 20 feet when convergence is beginning and divergence decreasing. The fact is that at a point 19 mm. in front of the line joining the centers of rotation, testing will show that adduction and abduction are equal.

Sursumvergence, or turning the eyes upward, involves contraction of the superior recti and the inferior obliques. The lateral movement to which the eyes are exposed by the action of the obliques is neutralized by antagonistic muscles and the corneæ are turned straight up.

Deorsumvergence, or turning the eyes down, involves contraction of the inferior recti and superior obliques, the lateral movements, as before, being controlled by their antagonists.

PART II.

STRUCTURAL ANOMALIES.

CHAPTER I.

OCULAR PALSIES.

OCULAR PALSIES are either of intracranial or of orbital origin. The intracerebral areas, invaded by such lesions, include the highest cortical centers,[1] the fibers connecting these centers with the oculo-muscular nuclei in the 4th ventricle,[2] the nuclei themselves,[3] and the trunks of the nerves to the ocular muscles anywhere from the 4th ventricle to their point of escape from the cranial cavity into the orbit. According to Fuchs, lesions of the nerve-trunks are due directly to degeneration of the nerve itself, or, indirectly, to interruption of its function by disease in its neighborhood, such as meningeal exudate, thickening of the periosteum, neoplasms, hemorrhages, and wounds; also by vascular changes, such as atheroma, aneurysm, emboli, and thrombi. The underlying cause is generally to be found in constitutional disease, preëminently syphilis. Strikingly causative in this class are tabes and general paralysis of the insane, the rheumatic diathesis, basal meningitis, and brain-tumors. Other conditions occasionally productive of ocular palsies are the various forms of intoxication, tuberculosis, diabetes, nephritis, diphtheria, influenza, hysteria, and many of the rarer central nervous diseases.

Orbital palsies arise from some local cause usually situated at the entrance of the nerve into the orbit through the superior orbital fissure, and are sometimes due to an affection of the nerve itself, at other times the result of pressure upon or disease adjacent to the nerve. There is a class of palsies not caused by influences acting in the orbit but on the nerve within the muscle, which, by Mauthner and some others, are termed "peripheral," and are ascribed to rheumatism, although Mauthner himself says that such palsies are not infrequently the forerunners of some serious central nervous disease, and are always to be regarded with suspicion. It is our own feeling that if many of these cases were followed out for a number of years until their real origin became apparent, we would hear less of "rheumatic" palsies.

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[1] The so-called "cortical palsy," Collins and Wilde. *Am. Jour. Med. Sci.*, vol. cii.

[2] The so-called "cortico-peduncular palsy," Collins and Wilde. *Am. Jour. Med. Sci.*, vol. cii.

[3] The so-called "peduncular or nuclear palsy," Collins and Wilde. *Am. Jour. Med. Sci.*, vol. cii.

Congenital ocular palsies may be:

1. Traumatic, from forceps delivery.
2. Inflammatory, affecting the nerve directly or indirectly.
3. Neoplastic, due to cranial or orbital tumors.
4. Teratologic, due to arrested development.

Ptosis is the most frequent variety of congenital palsy, with paralysis of the external rectus as a good second, and, strangely enough, contracture of the antagonists is not seen in this variety. Marina mentions, as quite frequent, conjugate convergence paralysis, the integrity of the internal recti being preserved. Mauthner and Dufour consider external ophthalmoplegia (paralysis of all the extrinsic muscles) as a nuclear disease; Mobius regards it as a special infantile disappearance of the nucleus, and recently Simmerling has found true disappearance of the nucleus with degeneration of some of its fibers; at times it is complicated with palsy of the facial nerve. Inability to turn the eye upward has often been found to coexist with congenital ptosis, and autopsies have shown absence of the superio rectus in many such cases. Perhaps similar defects lie a the bottom of many other congenital paralyses.

SYMPTOMATOLOGY.

The main symptoms by which ocular palsies reveal themselves are: 1. Limitation of movement; 2. False fixation; 3. Diplopia; 4. Vertigo; 5. Vicarious rotation of the head.

Limitation of movement in the direction of action of the affected muscle, is shown when a test object held in the median line is moved towards the side of the paralysis. The sound eye follows the movement easily as far as the limits of the field of binocular fixation, while the cornea of the squinting eye will be seen to stop at the median line; or, if it can be rotated further in that direction, it will be by a series of jerks and irregular contractions.

In a case of partial paralysis, the limitation of movement may be so slight as to be almost invisible. The globe, no longer under the control of six muscles, is rotated from the primary position to a secondary one, the resultant of the combined action of the five sound muscles. This deviation may be objectively determined roughly by inspection, and accurately by the use of the perimeter or Stevens's tropometer, and subjectively by the degree of prism necessary to displace the image of the affected eye from its false position back into the vertical or horizontal line, or to fuse it with the true image. As a matter of fact, very little information can be gained by observation of the limitation of movements, because all the eye-movements are under the control of more than one muscle. Therefore, paralysis of one muscle may be, in part, compensated for by the action of its synergist. Again, the arc of the circle of upward and downward rotation is normally much less than the lateral, and imperfect movement is less easy to detect in vertical than in horizontal palsies. The cornea is rotated by the sound muscles in the direction opposite to the affected muscle, the degree of rotation and forced immobility depending upon the completeness of the paralysis and the amount of secondary contraction of the antagonist muscles. This is known as "primary" deviation. If the sound eye is covered, it will be seen under the cover to have assumed the squint corresponding to that of the affected eye. This "secondary" deviation is more pronounced than the primary, since the extremely strong innervation necessary to stimulate the paralyzed muscle involves overaction of those muscles in the sound eye acting with it in associated movements. For the reasons given above, the rotation of the cornea cannot be observed with accuracy in the case of vertical paralysis, and we cannot derive much advantage from this measure.

False Fixation.—Objects in space cannot be accurately located with the squinting eye because their images do not fall upon the fovea, but upon a neighboring portion of the retina, in a direction and at a distance from the fovea depending upon the muscle paralyzed and the degree of the paralysis. The test instituted by v. Graefe, and called by him the "touch test," consists in closing the sound eye and at once requiring the patient to point towards a designated object. Instead of indicating the true position, the finger will point to a part of the field governed by the affected muscle. In cases of long standing, this test is worthless, because the patient has learned to use his judgment and to make allowance for false projection.

Diplopia.[4]—This is the most constant, persistent, and annoying symptom of all paralyses. Two images (the true and the false) of every object are seen in that part of the field governed by the paralyzed muscle. The discrimination by the patient of the true from the false image is not always possible, and other senses than that of sight must be called upon to aid in the discrimination. The physician is aided in his diagnosis by: (1) the relative position of the two images; (2) their relative movability when the test-object is carried in all directions; (3) the loss of parallelism of the vertical or horizontal axes of the two images, this loss becoming more marked in the periphery of the binocular field; (4) the conscious fixation, or the patient's testimony as to which is the true and which the false image. In some cases all tests fail.

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[4] It is assumed that *monocular* diplopia is not present.

Vertigo depends upon false projection and diplopia. It is most annoying in the early stages of the paralysis and with few exceptions becomes less and less troublesome as the case wears on. It disappears, promptly and entirely, upon excluding the deviating eye from the act of vision and, partially, upon excluding the sound eye. The mental disturbances are at times so great that the patient is discouraged from his usual pursuits, shuns society, seeking seclusion and darkness, and eventually may be driven by despair to self-destruction.

Vicarious Rotation of the Head.—In order to supply the function of the silent ocular muscle, the head is rotated by the action of compensating neck-muscles. By this means the false image may be made to fuse with the true over a much larger portion of the common field than if the head is held in the primary position and considerable diplopia and mental confusion are avoided. The secondary head position depends upon the muscle involved. In paralysis of an elevator, the eye is turned down and the head therefore thrown backward on its horizontal axis; if a depressor, the reverse; if the right externus, the head is turned to the right on its vertical axis; if the right internus, to the left; if the left externus, to the left; if the left internus, to the right. While these compensatory rotations do not specifically determine the muscle affected, they are valuable adjuncts to the diagnosis, particularly if the elevators or depressors are involved.

SPECIAL PARALYSES.

Paralysis of the oculo-motor is recognized by

1. **Complete Ptosis.**—The abnormally smooth upper lid lies over the ball unmoved by the levator, although it can be partly raised by forcible contraction of the occipito-frontalis. That the partial elevation is wholly due to the latter muscle can be readily demonstrated by pressing one's fingers upon the eyebrows, thus prohibiting action of that muscle, when the patient will endeavor to compensate for the lost action of the levator by holding his head far back.

2. **Deviation and Immobility.**—All movement of the ball, excepting that dependent upon the superior oblique (4th nerve) and the external rectus (6th nerve), is abolished, and the cornea is rotated directly outwards and possibly downwards. The physiologic action of the superior oblique alone is well shown in this paralysis. The effort made by the patient to turn the eye farther out and down results in slight actual movement down and out, but the upper end of the vertical meridian of the cornea will be plainly seen to turn toward the median line.

3. **Dilatation of the Pupil.**—The dilatation is moderate and is due to the paralysis of the circular sphincter pupillary fibers. Further dilatation can be obtained by the instillation of a mydriatic, which empties the blood-vessels of their contents, and possibly stimulates the sympathetic fibers and relaxes the choroid generally. The pupil is absolutely unresponsive to light admitted directly or consensually through communicating fibers from the 3d nerve of the opposite side, also to the stimuli of convergence and accommodation; (the response to the associated action

cannot, of course, be elicited in paralysis of the accommodation and internus).

4. **Failure of Accommodation.**—In emmetropia the inaction of the ciliary muscle (cycloplegia) consequent upon complete 3d nerve palsy does not diminish the previous acuity of vision for distance; in hypermetropia the amount of loss depends directly upon the degree of defect; in myopia of less than 1 diopter it has no appreciable effect. In estimating the near point, in E. and H. and in low degrees of M. the tests should always be made while the far correction is worn, or its kind and degree known and allowed for. The estimation of the loss is readily determined by restoring the reading power with a glass of known focal length and then measuring the distance from the patient's eye that the print is read. Thus in E. + 3 will allow reading at 13″; in H. of 2, + 5 would be necessary; in M. of 3 D. the test type would be held at 13″ (without — 3); in M. of 5, — 2 would restore the reading power at 13″.

5. **Diplopia.**—The false image is on the side opposite to the affected muscle, *i.e.* crossed and on a level with the true, excepting in the extreme periphery, when it may be slightly higher and tilted away from the true one. Wishart[5] states that when the image of the affected eye is straight and that of the sound eye tilted, nuclear lesion of the same side as the palsied eye may be diagnosed, because as the fibers of the 3d nerve diverge to the nucleus, a few of them pass through the raphe and cross over to the nucleolus for the inferior oblique in the opposite nucleus, and the inferior oblique of the affected eye thus escapes involvement while the inferior oblique of the sound eye is palsied. On the other hand, tilting of the image of the affected eye indicates a lesion of the *trunk* of the nerve,

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[5] *Jour. of Nerv. and Mental Dis.*, Dec., 1897.

and the inferior oblique of the affected eye suffers along with the rest of the 3d nerve muscles of that eye.

The images separate from each other the farther the test-object is carried toward the limit of the field governed by the paralyzed muscle. In paresis, the diplopia begins as soon as the middle line is crossed, but in total paralysis the field of diplopia passes beyond the median line, and, in old cases, attended with secondary contraction, single vision is unattainable. The slight elevation of the false image is caused by the contraction of the superior oblique. In the middle portions of the field the vertical turning of this muscle is concealed by the stronger action of the externus in turning the cornea outward; but when the abducting power of the latter is exhausted, the vertical action of the superior oblique becomes manifest.

RECURRENT OCULO-MOTOR PARALYSIS.

There is a somewhat unusual form of oculo-motor palsy, described first by Gubler in 1860, and later under the name of ophthalmoplegic migraine. It appears and disappears at irregular intervals from childhood to adult life, the paralysis generally increasing in severity and duration with each attack, until frequently, when the patient has reached manhood or womanhood, the palsy is complete and permanent. It is always unilateral and always affects the same nerve. The disease seems to affect both sexes equally, and the recurrences are preceded by migraine, vertigo, nausea and vomiting, and accompanied by concentric limitation of the visual field and lowering of central visual acuity. The etiology of the disorder is obscure. Most authorities ascribe it to a basal origin, although it is not inconsistent with the symptoms to assume a disturbance in the vascular supply of the 3d nerve nucleus in the anterior part of the floor of the 4th ventricle. It is not amenable to any known treatment.[5]

Recurring and partial ophthalmoplegia interna is characterized by partial or complete paralysis of the iris and ciliary muscle. For a period of days or weeks the pupil is dilated and unresponsive to light and the stimulus of associated action, and the accommodation is in abeyance. The symptoms entirely disappear, to recur at irregular intervals. The disease is probably nuclear in its origin and dependent upon variations in the vascular supply to the floor of the 4th ventricle. Treatment other than local applications of eserin seems to have no influence.[6]

PARALYSIS OF INDIVIDUAL BRANCHES OF THE MOTOR-OCULI.

Internal Rectus.—Inward movement of the cornea past the middle line is limited or abolished. The face is turned towards the opposite side, the diplopia is crossed and is found in the field governed by the paralyzed internus, in which direction the false image separates more and more from the true one. The two images are on a horizontal line over the major part of the diplopsic field. At the extreme periphery the false is slightly higher than the true image, and its vertical meridian is tilted away from it. For example, paralysis of internus of the right eye: the cornea is turned outward and cannot be rotated inward past the median line; rotation in all other directions unrestricted; the false image is to the left of the true, on a level with it, and separates from it the farther the test-object (candle-light) is carried to the left. In order to compensate for the abnormal divergence of the eye, the face is turned to the left on its vertical axis; diplopia commences in the middle line; or, in old cases, where secondary contraction of the externus has set in, diplopia is seen over the entire field. Paralysis of the internal rectus, unassociated with palsy of some or all of the others supplied by the 3d nerve of this same side, or with the opposite internus (paralysis of convergence), is extremely rare. The lesion may be a change in the muscular structure itself, congenital, idiopathic, traumatic, or due to an exceedingly fine nuclear disease.

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[5] Philip Coombes Knapp brought together the histories of forty such cases in the *Boston Medical and Surgical Journal* for 1894 (pp. 308 to 312), since which time several others have been reported.

[6] This disease was first described by Hansell in *The Ophthalmic Record* for April, 1898.

Superior Rectus.—When the gaze is directed upward, the sound eye follows the test-object, but the cornea of the affected eye stops at the horizontal line and the efforts to continue its upward turning result in divergence. Diplopia is found in the upper half of the field of vision. The false image moves farther from the true as the light is carried upward, the former indicating the affected eye by its more rapid movement and greater elevation. Its upper end is tilted towards the temporal side. The head is tilted backward on its horizontal axis. In diagnosing paralytic from functional disease of the superior rectus, the extent of the field of diplopia is an important feature. In paralysis, the false image leaves the true below or at the horizontal line. In functional disease, diplopia is common only in the periphery of the field and does not commence until the object is moved far above the horizontal line. Again, in paralysis, efforts to turn the eye upwards result in a rotation upwards and outwards. In functional disease, the limitation of rotation is not manifest, and can be determined only by careful measurement with the perimeter or some other special instrument. The onset of paralysis is sudden; it is preceded or accompanied by headache of a few hours or days, and may be associated with symptoms of some general disease. A history of traumatism or disease of the nervous system may be elicited. In functional disease complaint is seldom made of diplopia, and the patient will probably give the history of long-continued headache, asthenopia, and reflex nervous symptoms.

Inferior Rectus.—The movement of the eye downwards is limited, especially in abduction. The eye is diverged and the diplopia is in the lower half of the field, the lower image belonging to the affected eye, with its upper end tilted towards the temple. As the eye is adducted, the distance between the images decreases. In abduction the false image becomes more tilted, the tilting diminishing when the object is moved to the side opposite the affected eye.

Inferior Oblique.—The elevation of the eye is limited, especially in adduction, although no deviation of the eye is apparent. Diplopia is in the upper half of the field, the false image, tilted temporalward, is above the true and on the side of the diseased eye.

For the prognosis and treatment of paralysis of the oculo-motor nerve, and of the individual muscles under its control, see the last two paragraphs in this chapter.

TROCHLEARIS PALSY.

A patient presenting paralysis of the right superior oblique muscle will complain of diplopia on looking down. It will be found that the lower image which belongs to the affected (right, for example) eye, is inclined to the left, and that the vertical distance between the images increases as the eye is depressed and adducted. Abduction will increase the obliquity of the image. The limitation of movement of the eye will become apparent when extreme down and inward rotation is attempted, and on looking down the patient will see things lower than they ought to be. If palsy of the superior oblique muscle occurs as an isolate paralysis, the patient can, by inclining the head downward and to the right or left, produce fusion of the images ; an if the palsy be a transient one, it may thus never come t the notice of the surgeon. However, isolated trochleari palsy is infrequent, and as a focal symptom is even mor uncommon. When the trochlearis is palsied, it is usuall in conjunction with other muscles of the 3d nerve group.

The features of individual paralysis of the superio oblique are well shown in the following history taken fron our records :

Case I.—March 6, 1896. J. M., aged 43. For thre months he has had diplopia—one image above the other V. R. $\frac{20}{50}$, partly, L. $\frac{20}{200}$; low myopia and astigmatism ; ful vision with correction ; no disease of the media or eye grounds. Covering the right eye with the blue and th left with the red glass and testing with the candle-flame the red light separated from the blue in the lower field, th upper end tilted towards the blue, always homonymous i the left field, the distance between them increasing th farther the candle was carried down towards the right When the image had reached 20° to the right, the fals image became crossed over, and when carried to the left i crossed at 30°. The movement of the eye down and i was limited. The diplopia when first noticed was vertical A prism of 6 base down, combined with 1° base in, i front of the left eye, fused the images in the median field The diagnosis in this case rested between paralysis of th inferior rectus and paralysis of the superior oblique. Tha is, since downward movement of the left eye was limited evidently one or both of the depressors must have bee affected. The normal action of the superior oblique is t

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[1] Several instances of this associated paralysis are to be found in Marina's monograph on the ocular palsies.

turn the cornea down and out, with the upper end of its vertical meridian tilted inward; of the inferior rectus, to turn the cornea down and in, with the upper end of its vertical meridian out. In paralysis, therefore, of the superior oblique, the eye would be turned in rather than out, and the images would be homonymous, as in the above case. In paralysis of the inferior rectus, movement down and in would be limited and the images would be crossed. In both cases the false image would be tilted towards the true. The lesion, in this case, may be basal or nuclear. It is impossible to tell exactly its character, since the paralysis remained an isolated one without complications. The patient had a syphilitic history, but did not improve under large doses of iodid of potassium and bichlorid of mercury, nor did the addition of strychnin improve the symptom.

ABDUCENS PALSY.

The long course of the 6th nerve at the base of the brain renders it peculiarly liable to arrest of function during disease, or from traumatism to the skull. Indeed, the external rectus leads in the order of frequency of individual ocular palsies, the order being external rectus first, then unilateral oculo-motor palsy, paralysis of the superior oblique, the inferior rectus, the superior rectus, the internal rectus, and the inferior oblique. Duane[8] would award to the superior rectus second instead of third place. The subject of this affection will complain principally of constant diplopia, homonymous in character. The distance between the images will increase as the test-object is carried to the right if the palsy is right-sided, and to the left if the left external rectus be affected. Convergent strabismus will be simulated as the eyes are moved toward the palsied side, and the face will be turned toward the affected muscle. Secondary inward deviation of the sound eye is a late symptom.

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[8] Knapp's *Arch. of Ophthal.*, 1894, p. 61.

Abducens palsy is commonly seen after fracture, or accompanying a syphilitic or other inflammatory process at the base. It also occurs with diseases of the pons. Peripheral or orbital abducens palsy, when not rheumatic in character, is said by Wood to indicate syphilis in adults and tubercular disease in children. However, in the latter connection, it must not be forgotten that abducens palsy in children is not infrequently congenital, and may also be traceable to forceps delivery. (Marina.)

If paralysis of the external rectus is not peripheral, basal, or pontine, the lesion resides in the nucleus, the cortex, or the connecting tracts, in any of which cases, if the origin be not specific or traumatic, the prognosis is highly unfavorable and ominous of evil for the near future.

The treatment of this particular palsy differs in no wise from that of the other ocular paralyses, outlined in the general chapter on treatment.

The following instance of isolated paralysis of the external rectus is taken from our records:

Case I.—January 30, 1896. J. S., aged 65, widower, engineer of a shifting or yard engine. Has always been an unusually healthy man, no subjective or objective testimony of rheumatism, syphilis, or tabes. Four days ago, while at work on his engine, objects began to appear double, and the confusion became so great within an hour that he was compelled to quit work. The anterior ocular segment of both eyes was normal; pupils 4 mm., and regularly round; V. O. D. and O. S. $\frac{5}{36}$. With + 1.00 D. S. = $\frac{5}{6}$, ocular movements were smoothly and completely executed in all meridians save in movement to the left, when the left rectus externus refused to carry that eye more than 5 mm. beyond the median line.

The ophthalmoscope showed clear media and normal fundi. With a red glass before the right eye, there was shown homonymous double vision, the distance between the images increasing as the candle was carried to the left. There was no upward or downward displacement of the false image, nor was it tilted. A 30° prism, base out, before the left eye fused the images at 4 meters; but the patient, even by the fourth day, had learned that by turning his face toward the left he could fuse images and avoid diplopia. Patient was ordered $\frac{1}{16}$ grain biniodid of mercury, and strychnin in increasing doses three times a day. Urinalysis, made by his general medical adviser, shows neither sugar nor albumin, but reveals an excess of uric acid.

Four days later, fuses images with 11° prism; 28 days later, fuses images with 5° prism.

Case did not return for further observation. The fact that the patient had been a railroad man all his life, that the palsy was in all likelihood non-focal, and that it disappeared so rapidly under biniodid of mercury, would indicate that the foregoing was an instance of specific abducens palsy.

CASE II.—Abducens paralysis due to traumatism of the skull. M. J., aged 25, applied at the Polyclinic Hospital, December 15, 1897, complaining of constant diplopia. He stated that on December 8th, while skating, he fell and struck the left side of his head against the ice. He was dazed for a short time, but recovered and noticed nothing unusual until evening, when objects became double and remained so. The right eye was converged; the cornea could not be moved past the median line; there was homonymous diplopia on the right side of the field. Media and eye-grounds normal. The lesion was probably basal and consisted of minute hemorrhage either in the sheath or pressing upon the trunk of the 6th nerve on the right side. No cerebral symptoms developed and there were no other complications. This, then, is an instance of traumatic paralysis occurring in the long course of the 6th nerve, at the base of the brain, from hemorrhage due to traumatism of the left side of the skull. The final outcome of the case is unknown.

Inasmuch as the nucleus of the 6th nerve is said to be the center for conjugate deviation, a few words may here be said of

CONJUGATE PALSIES.

Inability to move the eyes in association to the right or, as the case may be, to the left beyond the median line, is sometimes seen in individuals who nevertheless converge close up to the ordinary convergence near point. This paralysis of synchronous action of one internal rectus and the external rectus of the other eye is known as conjugate palsy, and may follow upon lesions in the cortex, the corona radiata, the internal capsule, or the pons. It is not an uncommon symptom in gross lesions of the cerebrum and the pons, although the conjugate palsies secondary to cerebral lesions are exactly the reverse of those seen in diseases of the pons. Swanzy[9] contrasts the association of the palsies with their causal lesions as follows:

Cerebral lesions.	Destructive. Eyes turned away from palsied side.
	Irritative. Eyes turned toward convulsed side.
Pontine lesions.	Destructive. Eyes turned toward palsied side.
	Irritative. Eyes turned from convulsed side.

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[9] Diseases of the Eye, fourth edition.

Wernicke[10] speaks of conjugate palsy of upward and downward movement of the eyes as being secondary to disease of the corpus striatum and of the optic thalamus.

PARALYSIS OF CONVERGENCE.

CASE I.—A woman, aged 24, was under the care of Dr. Dercum, at the Jefferson Hospital, on account of inability to walk, because of loss of coördination of the extremities. Examination revealed that the eyes and their appendages were normal in every respect, with the exception that the patient had lost absolutely all power of convergence. She could follow with both eyes a test-object moved to the right, left, above, or below, the optic axes remaining parallel throughout all these movements; but when the test-object was moved in the median line from the distant to the near point, she altogether failed to follow the movements with either eye. She had no diplopia in any direction, and no ocular paralysis. The optic axes remained parallel in all movements, hence the image of the test-object fell on corresponding parts of each retina. The symptom probably arose from cerebellar disease.

CONJUGATE VERTICAL PARALYSIS.

CASE II.—March, 1895, J. B., aged 35, complained of diplopia. Vision, right $\frac{20}{30}$, left $\frac{20}{30}$; left hypertropia 6°, exotropia 4°, from paralysis of the right superior oblique. There was, in addition, *complete paralysis of upward movement of both eyes.* An object held above the horizontal line could not be followed by either cornea, and efforts to look up resulted only in partial contraction of the levator palpebræ. In April, 1897, divergence and upward deviation of the left eye marked; vision of the right had fallen to

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[10] Nervous Diseases, by Dercum.

$\frac{20}{40}$, but remained full in the left; ophthalmoscope showed commencing atrophy of the right optic nerve; in the left retina, the arteries were smaller than normal, but not markedly so; both fields concentrically contracted for white and colors, right decidedly; pupils responsive to light and accommodation, but sluggish. In October, 1897, vision in both eyes fallen to $\frac{20}{50}$; left eye diverged and rotated slightly downwards; upward movement abolished as before, downward movement limited; optic nerve atrophy had further advanced. There was also incoördination of extremities, and walking was, on this account, difficult. The knee-jerks were exaggerated. This is a case of advancing ophthalmoplegia and optic nerve atrophy, the symptoms of which were preceded, for some years, by loss of upward movement in both eyes. The patient had no history of specific infection or constitutional disease.

PROGNOSIS.

Prognosis in ocular palsies hinges on whether the paralysis be: 1. Acute or chronic. 2. Individual or associated. 3. Traumatic or idiopathic.[10] 4. Due to a central or peripheral lesion.

It is further influenced by the nature of the general disease-process of which it may be a symptom.

An acute individual palsy, if traumatic or peripheral, offers in many cases a reasonably hopeful outlook. Especially is this the case if the patient is the subject of a traumatism, or of syphilis, rheumatism, or tabes. In the latter case, the palsy is likely of a peripheral nature, and though the prognosis as to the paralysis itself is favorable, it is important to remember that a transient external ocular palsy may be an early and significant symptom of locomotor ataxia, and should never be disregarded. Acute palsies of higher origin (basal, nuclear, or fascicular) are to be regarded with apprehension; and though they often prove transitory, they more frequently portend intracranial mischief—syphilitic, hemorrhagic, inflammatory, or neoplastic in character—and thus become symptoms of great value in estimating the probable nature of obscure central lesions.

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[10] Idiopathic, here used in the sense of a paralysis occurring without demonstrable lesion in a previously healthy person.

An acute associated paralysis, or conjugate palsy, while of itself of no special importance, and though not infrequently transient, is like the foregoing, ominous for the future, and should invariably lead to thorough-going investigation of the functions of the patient's central nervous system. It is occasionally hysteric in origin.

Chronic paralyses, of whatever character, are gloomy affairs, promising little for the present and less for the future. If the palsy be an individual or isolated one, it is apt to be accompanied by secondary contraction of the antagonist muscle of the same eye. Some people live through a long and busy career with palsy of one or more of the extrinsic eye-muscles, and suffer little or no discomfort therefrom after the acute stage is passed, but this is exceptional. Oftener some grave nervous malady unfolds its features, and the case drags its weary length along, or in the midst of life the patient may be in death. Such sudden termination is the rule in paralyses occurring in arterio-sclerotic individuals.

TREATMENT.

As to treatment, ocular palsies arrange themselves in two classes: those in which the origin is more or less apparent, offering opportunity for rational treatment, and those in which the obscurity surrounding the case compels empirical therapy.

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Fortunately for both surgeon and patient, this phase of the subject is much simplified by the aid of statistics, which show the predominant influence of syphilis in the production of paralyses of the ocular muscles. According to Alexander (*Syphilis und Auge*), 59.4 per cent. of all ocular palsies are specific, the oculo-motor being most frequently selected by the disease. Vigorous anti-syphilitic medication is, therefore, indicated in all ocular palsies that are not traumatic, or cannot be shown to be distinctly rheumatic, tubercular, or gouty; and the caprice of the surgeon will largely dictate what shall be the mercurial or iodin preparation used, likewise the mode of its application. Moreover, mercury or iodin will be preferred according as the palsy is a secondary or tertiary syphilitic phenomenon; and it is well to bear in mind that all medication aimed at ocular palsies (and specific treatment in particular) is more likely to be fruitful of results if assisted with Turkish baths or such other measures as will promote increased functional activity of the skin. By this means most heroic internal treatment will often be generously tolerated.

Isolated ocular paralyses occurring in adult life in an apparently healthy middle-aged individual, should invariably arouse suspicion of, and thorough search for, tabes, which, if the suspicion be confirmed, calls for anti-syphilitic treatment in fully 90 per cent. of all cases (according to Erb and Fournier). Every such suspect deserves careful interrogation of the pupil reflex to light and accommodation, and of the patellar tendon reflexes.

Essential rheumatic palsies (a class that is yearly growing proportionately smaller) yield generally to salicylates or iodids, the former in the acute, the latter in the chronic forms of the disorder. Other rheumatic remedies have been favorably mentioned from time to time.

When paralysis of an ocular muscle is the result of encroachment of a tubercular focus upon the nucleus or trunk of the nerve supplying the palsied muscles, treatment is of little avail; happily, such conditions are rare.

Palsies of gouty origin are commonly the peripheral manifestations of an obliterating endarteritis or hemorrhage in that part of the floor of the 4th ventricle, in which the 3d, 4th, and 6th nerve nuclei are located. If the arterio-sclerosis be not too advanced, much benefit, and even complete recovery, may follow upon the active use of absorbents and diaphoretics, at the same time guarding well the heart with digitalis or strychnin. This treatment is also applicable to the palsies associated with the emboli and thrombi common to cardio-vascular disease.

The treatment of palsies secondary to fracture of the cranium, either in the fronto-motor region of the calvarium or at the basis cranii, is embraced in the general treatment of the fracture itself, namely, rest in bed, antiphlogistics and absorbents, or surgical interference. The same management holds good for the palsies seen with orbital or sphenoidal fractures. If the nerve trunk or trunks be lacerated, treatment will accomplish little, while antiphlogistics and absorbents will go far toward restoring function if its arrest be due to the pressure of hemorrhage or inflammatory exudate. For those palsies that accompany inflammations or tumors of the brain and its membranes, the treatment must be aimed at the major affection. If the process be a specific one, its therapy is simple. If it prove tubercular, rheumatic, gouty, arterio-sclerotic, senile, or neoplastic, symptomatic treatment only is called for, and will often prove unavailing.

There remain to be mentioned, in the therapy of these conditions, strychnin and electricity.

The value of strychnin in restoring palsied ocular muscles has, perhaps, been over-estimated. This may be due, in part, to the fact that it has been exhibited indiscriminately in all forms of palsy, whether cortical, fascicular, nuclear, or peripheral. It is in the latter class only that it is of marked value, although its use is admissible in the acute stages of almost any palsy. A method that is highly esteemed is to order 1 gr. of strychnia in 1 oz. of water, beginning with a 10-drop dose (gr. $\frac{1}{48}$), 3 times daily, and increasing the dose 1 drop each day until toxic symptoms appear.

As to electricity, faradism is most popular, although Fuchs is opposed to the induced current. A strength of 1 mp. should never be exceeded. If, as some prefer, the slowly interrupted constant current be used, 3 mps. is all that will usually be borne with comfort; the cathode to be used over the closed lid, and the anode (or indifferent pole) over the corresponding temple or mastoid. The quantity of current that really passes through the affected muscle is uncertain. Some surgeons prefer to place the anode on the sclera directly over the palsied muscle after cocainizing the conjunctiva.

It is ofttimes necessary, in addition to the medical treatment of the paralysis itself, to relieve the annoyance which the diplopia and vertigo bring with them. If the palsy be slight, amounting only to a paresis, prisms which fuse the images and are properly placed in an ordinary spectacle frame may be worn, and the patient thus rendered comfortable until such time as the cure may be accomplished. When the diplopia cannot be thus overcome, it will be best to exclude the paralytic eye from all efforts at binocular vision, by placing before it, in a spectacle frame, a piece of ground glass.

In practising the mechanical treatment suggested by Michel, the eye is cocainized and the conjunctiva seized near the insertion of the palsied muscle, when the eyeball is drawn forcibly as far as possible beyond the limit of contraction and then back again. Michel recommends daily use of the movements for about a minute each time.

In long-standing paralyses, operation offers the best results. Section of the antagonist, combined with advancement of the affected muscle and adjoining capsule and conjunctiva, affords the latter the most favorable mechanical conditions for work. This presupposes, however, some slight degree of contractility; consequently, complete paralyses are incurable by operation.

PART III.

FUNCTIONAL ANOMALIES.

CHAPTER I.

HETEROPHORIA.

Section I.—GENERAL CONSIDERATIONS.

In the study of the relation of the muscles to each other in states of abnormal tension and relaxation (but without paralysis), we leave the clearly defined field of recognized symptoms and conditions, and enter that of uncertainty, vagueness, and speculation; we pass from the logical results of anatomic changes to the confused manifestations of unknown psychologic disturbances. We do not deal with demonstrable lesions of the muscular or nervous system, but with *tendencies* to incoördination, the result of innervational peculiarities that thus far have been untraceable by the dissector's knife or physiologist's microscope. We are brought face to face with the ocular complications of disordered health, of inherited or acquired abnormal susceptibility, of illogical and uncommon results of common causes, of individual idiosyncrasies—in a word, of unbalanced nerve-action. While it is not disputed that functional muscular anomalies follow known causes according to known principles of cause and effect, it is maintained that some of the most difficult problems offered for solution cannot be explained by any satisfactory theory. Obstacles surround the examination and treatment of every case and can only be overcome by repeated trials with modern diagnostic methods and the persistent applications of physiologic and conservative remedies. The *individual* element must not be lost sight of, and every factor, ocular or extraocular, that can have a bearing on the solution thoroughly investigated. The oculist is not the refractor or the tenotomist alone, but is the physician as well.

Functional deviations are so intimately bound up with the refractive status as to compel frequent mention of the latter in discussing the various forms of muscular imbalance. Indeed, no problem in heterophoria is free from the complicating influence of the action of the ciliary muscle, whether the eye be emmetropic or not. The importance of this relation is so great that some high authorities maintain to-day that all muscular "imbalance" (Gould) proceeds from errors of refraction, and that the persistent wear of correcting lenses will ultimately dissipate the heterophoria. Functional deviations of the eye depend, further, for their solution upon heredity, age, sex, social condition, temperament, physique, vocation, hours of work, mental worry, and numerous other minor factors. It will thus be readily seen that they are not so simple in their cause and treatment as has been suggested by some authors. Many of the older writers looked upon such deviations of the ocular muscles as symptoms of a lowering of the general nervous tone (whims of the nervous system, as some have called them), or of some general pathologic process; and although later methods and up-to-date instruments of precision have plainly shown that a goodly number of heterophorias are not symptomatic but are substantive conditions attended by a train of their own symptoms, the view of these older writers was not without foundation, and it deserves particular emphasis in this day of exceeding specialism.

Muscular Balance.—The eyes may be said to be swung and held taut in the orbit by a delicate muscular harness. The four recti muscles in combination tend to draw the globe backwards into the orbit, which action is opposed in greater or less degree by the tendency of the superior and inferior oblique to bring the globe forward. Hence, a light degree of contraction of all the muscles is necessary o maintain the globe in the proper position during all vaking hours. This process, simultaneously active in oth orbits, produces coördination of the visual axes, so hat when the eyes are directed toward an object, their xes will meet exactly in that object, and the eyes are said o be balanced, or in equilibrium.

It is important to remember in this connection that the irection of the visual axes when the eyes are in the *anaomical* position of rest, (resulting from the form of the orit, the insertion of the optic nerve and the natural length f the muscles when not innervated), is generally divergent, arely parallel, and hardly ever convergent. But the funcion of normal eyes never allows independent deviation ; ence the parallel direction demanded for distance can ever be abandoned whilst we are awake, and the unconcious innervational habit pulls the eyes from the *anatomcal* position of rest to parallelism, which is the *functional* osition of rest ; *functional*, because the function of the yes has produced it, and *position of rest*, because the position assumed by habit and unconscious innervation is free rom all exertion.

Any disturbance of the factors just mentioned will ause muscular imbalance, and yet want of muscular balnce is not incompatible with perfect binocular vision, for, n many cases, visual axes that tend to deviate are brought ack to parallelism by increased innervation to a given nuscle or group of muscles, and it is this necessary extra utlay of nervous energy that frequently brings on asthenpia in small degrees of muscular imbalance. Functional isorders of coordination are manifest or latent. There nay be an *actual turning* of one optic axis from the other, nvolving a loss of associated movement, or the want of oordination may show itself as nothing more than a *ten-*

dency to deviation. Between these two extremes of want of equilibrium (the one representing the accomplished strabismus and the other latent overaction or underaction of synergist muscles) is a middle stage, characterized by the development of either the deviation tendency or the squint, according to the demand made on the different muscles; for example: the insufficiency of the internal recti for near work, described by v. Graefe, thirty years ago. The study of this subject will be simplified if the student will bear in mind that functional want of equilibrium concerns in all cases the destruction of the normal relation between two sets of opposing muscles rather than the insufficient or overaction of any one muscle. Thus, in esophoria we are dealing with the power of convergence, and not the strength of the internal recti. It is unscientific to attempt to separate the muscular from the innervational apparatus, the more so, as the latter comprises both the fusion-force and the impulse conveyed by the nerve-trunks; and equally futile is it to speak of one muscle as having more power, as measured by prisms, than its corresponding muscle in the other eye.

The various gradations from the slightest tendency of the optic axes to turn from equilibrium to the highest degree of permanent squint, are but steps in the same direction, or degrees of the same affection, and, in great part, the causes which underlie the one grade are found to produce all the others. For instance: a tendency to turn the visual axes towards each other has, as its fundamental cause, hyperopia. The degree of inward turning or tendency is determined largely by the personal equation. Many hyperopes have absolutely no want of equilibrium, and yet others, even with low grades of hyperopia, develop marked muscular disorders. It has been contended that because hyperopia does not produce a squint in every instance the squint must have some other cause, but it is well known that the same cause produces different effects according to individual characteristics. A low grade of hyperopia in a nervous or overwrought disposition will give rise to serious reflex neuroses, whereas the same degree of hyperopia in a constitution, robust, strong, and resisting, will have no symptoms whatever. Again, a high grade of hyperopia is, in some, perfectly consistent with equilibrium of the muscles, while, in others, it is the cause of decided strabismus. This individuality can only be described in vague terms. It is known as a neurotic disposition, an enfeebled, unresisting nature and susceptibility to disease, an exaggeration of reflex excitability, etc. Therefore, when all circumstances are favorable, reflex neurosis may arise from muscular anomaly, and there is no doubt that, in a few instances, the claims made by Stevens and Ranney are justified; but such cases are exceptional in even the largest experience, and the theory that a considerable proportion of the insane, of the epileptic, of the choreic, owe their disease wholly to the existence of either a refractive error or a consequent muscular anomaly, is dangerous and unsound.

In examining patients who have no organic disease of the eye or its environment, but suffer from reflex headaches and annoyances of various kinds, estimation of the condition of refraction and of the ocular muscles should be among the earliest findings. The investigation of the muscular status should be made both before and after mydriatics are instilled, because of the direct influence these drugs have in many cases upon both the kind and degree of the defect.

The nomenclature of anomalies of the ocular muscles has received a decided impulse in the past few years. For functional deviations Stevens has suggested a classification that has met with quite general acceptance in America and England. It is as follows:

Orthophoria, perfect binocular balance.

Heterophoria, imperfect binocular balance. Heterophoria presents many varieties; to wit:

Hyperphoria, a tendency of the visual axis of one eye to deviate above that of the other.

Hypophoria, a tendency of the visual axis of one eye to deviate below that of the other.

Exophoria, a tendency of the visual axes outward.

Esophoria, a tendency of the visual axes inward.

Hyperexophoria, a tendency of the visual axis of one eye to deviate upward and outward.

Hypoexophoria, a tendency of the visual axis of one eye to deviate downward and outward.

Hyperesophoria, a tendency of the visual axis of one eye to deviate upward and inward.

Hypoesophoria, a tendency of the visual axis of one eye to deviate downward and inward.

It must be remembered that, in the functional anomalies under discussion, both eyes are involved and that while the above nomenclature may describe the symptom it does not locate the lesion. For instance: right hyperphoria means either that the right elevators are too strong for the right depressors, or that the left depressors predominate over the left elevators. In other words, it simply signifies that one eye tends to turn upward or the other downward, without indicating which is the faulty eye.

To this classification, Savage would add cyclophoria, or insufficiency of the oblique muscles; and Duane, hypokinesis—deficiency of action of an individual muscle; hyperkinesis—excessive action of an individual muscle; and parakinesis—irregular action of an individual muscle.

SECTION II.—GENERAL DIAGNOSIS.

The purpose of the various tests for the determination of muscular imbalance is to so change the retinal image of one eye, by alterations of color, shape, or position, that the fusion-impulse will no longer be exercised, and that the tendency to deviation will become changed from a latent into a manifest one. Those which deform but do not displace the image from the fovea to some extra-foveal region, will most truly and accurately make manifest latent muscular error. They are, moreover, free from the suspicion of *creating* deviations of the visual axes by necessitating a comparison of the function of the foveal region of one eye with that of the retinal periphery of the other. Such tests are the Maddox rod, single or compound (preferably the latter), a strong cylinder from the test-case, a strong spherical lens, through which rays are admitted only in a small area around its center, and the cobalt blue glass with plane surfaces. The classic test, suggested first by v. Graefe, and adapted to accurate and speedy measurement by Stevens, is the prism test. A prism, with its base in, down, or up, too strong to be overcome by the muscles it antagonizes, will produce insuperable diplopia. For instance, to test lateral equilibrium, a prism of 8° held base down before the right eye, with its base-apex line exactly vertical, will refract the rays entering that eye into the lower portion of the retina, and a false image of the light will be projected into the upper part of the field. In orthophoria, the true and false lights are in a vertical line. In esophoria, the upper image will be to the right of an imaginary line running vertically through the lower or true light. The prism displaces the image of the light into the lower and, if the eye is turned in, also to the inner half of the retina; it is projected, accordingly, up and to the right. In exophoria, with the prism in the same position before the right eye, the false image will be above and to the left. The distance that the upper light is to the right of the imaginary vertical line, is the *linear* measure of the degree of the deviation. The prism (with its base *out* for esophoria, *in* for exophoria) necessary to move this image into the vertical line will be the angular measurement of the deviation. The principle of this test is exactly the same as that of all others, namely, the destruction of the unconscious fusion-impulse. The individual seeing two images of the same object contemporaneously, accepts the impression that the two images represent two objects. Fusion force having been thus destroyed, the deviation *tendency* becomes an *actual turning*, and the eyes assume that position which is most restful for them and to which they are impelled by the conditions present in the individual case.

THE METHODS OF DIAGNOSIS.

1. The Prism Test.—Diplopia is induced by a prism strong enough to prevent fusion, held with its base down, up, or in, before one eye. The prism commonly employed is of 10°. This strength is sufficient, not only to produce insuperable diplopia, but to separate the false from the true image by an interval great enough to allow slight deviation tendencies to cause a lateral displacement of the images that is instantly perceptible. Two serious errors arise when higher degree prisms are used, namely, the false image is refracted to a point on the retina so far removed from the real fovea, that the findings may not accurately represent the anomaly; and the slightest turning of the prism from its axis will vitiate the result by producing artificial heterophoria. In Stevens's phorometer, two prisms of 4° each are held in a frame that can be rotated to test either th horizontal or the vertical tensions. The instrument is cor venient, because both vertical and lateral imbalance can be determined by simply revolving the test-prisms into the horizontal or the vertical meridian. A total of 8° is selected, because, at the distance at which these prisms are placed from the patient's eyes, muscles of average diverging power cannot fuse the double images. (See Fig. 3.) With the instrument fixed to test the lateral muscles, two images will be seen. In orthophoria these images are in a vertical line, for although single vision has been destroyed, no hitherto latent tendency to turn the eyes in or out has become manifest, and the lateral muscles are said to be in equilibrium. In esophoria or in exophoria, one light, instead of being exactly above the other, will assume a position to the right or left, because the eyes are now free to respond to the influence of the relatively stronger act—convergence in esophoria and divergence in exophoria. The degree of prism now necessary to bring the upper image directly over the lower one, is the measure of the deviation. It must be remembered that we are dealing, not with one, but with both eyes. For instance, in esophoria we study *not one internus*, but the power of con-

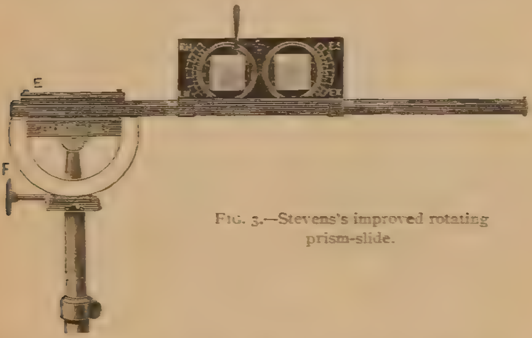
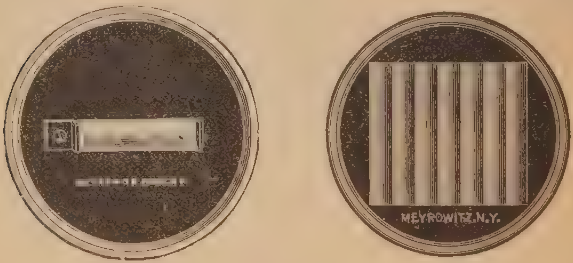


FIG. 3.—Stevens's improved rotating prism-slide.

vergence as contrasted with the power of divergence, and by this test we determine in esophoria that convergence is abnormally strong.

What is practically the same test may be conducted (as above mentioned) by holding a strong prism, base up or down, before either eye and measuring the deviation of the second light from the vertical line by a prism, base in or out, before the other eye. However, in our opinion, the prism test does not always indicate the true condition, for the reason that the foveal region in each retina is not considered, but rather the fovea in one eye and a point outside the fovea in the other eye. Hence, while the fusion-impulse is destroyed (which is necessary), we are expecting identical muscle-action as the result of stimulation of different retinal areas in the two eyes. We have found that this test shows exophoria to be more frequent than esophoria, and have often found a positive contradiction between the results of this test and those obtained by the use of the Maddox rod and other tests.



a. *b.*

FIG. 4.—*a.* Maddox simple rod.
b. Maddox compound rod.

2. **The Maddox Rod** (Fig. 4) consists essentially of a rod of glass conveniently adjusted in a disc to fit the trial frame. When light is refracted by a glass rod, a luminous point

becomes a line or streak, since the rod is nothing more than a very strong cylinder. If this rod is held vertically before one eye, the image of the light seen by that eye is a horizontal streak; if held horizontally the image is a vertical streak. In orthophoria the candle-flame is seen as a streak by the eye before which the rod is placed, the streak passing through the unaltered flame seen by the other eye. In heterophoria, the streak is seen either to the left, to the right, above or below, according to the nature of the defect. In esophoria, the streak is on the same side as the eye which sees it. For example, a Maddox rod is so held before the right eye, that the streak is vertical: it will be to the right of the candle-flame. In hyperphoria, however, the phenomenon is a little more confusing. For instance, in right hyperphoria, if the rod is placed before the right eye, the streak will be seen below the flame; if placed before the left eye, it will be seen above the flame; in left hyperphoria, the rod before the left eye will produce a streak below the flame; before the right eye, a streak above the flame. The prism necessary to change the position of the streak from its faulty position into the light (the orthophoric position), is the measure of the heterophoria. This instrument is simple, inexpensive, accurate and trustworthy, especially in its latest form of the compound rod. The advantage of the latter over the simple rod is that only ordinary care in its adjustment is necessary. With the simple rod it is essential to accuracy that the rod shall be in line with the visual axis. While the image is greatly distorted by this test, yet the foveal region of the retina is included in the site of the image, and we are studying the relations of the two foveas to each other, and not, as in the prism test, the relation of one fovea with the circummacular portion of the other retina. By this test we find esophoria more common than exophoria, and from the study of other conditions, such as the refraction, are led to believe that this is the true relation. For the relative positions of the streak and light in the various muscular states, see Fig. 5.

3. The Convex Spherical.—A convex glass of the strength of 10 to 20 diopters, covered in all parts excepting its center, is held before one eye. The image seen by that eye assumes the shape of a large blur upon the fovea and the adjoining retina. In orthophoria, the clear light seen by the uncovered eye will be situated in the center of the blurred image of the covered eye. (Fig. 6, p. 65.) In heterophoria of low grade, the clear image appears on the outskirts of the blurred image, and in the higher grades will be separated from it. The interval between the two is the measure of the grade, and the prism required to refract the clear image into the center of the other will be the prism measurement of the heterophoria. This test can be accurate only when but a small portion of the exact center of the lens is left clear, because rays of light passing through it at any con

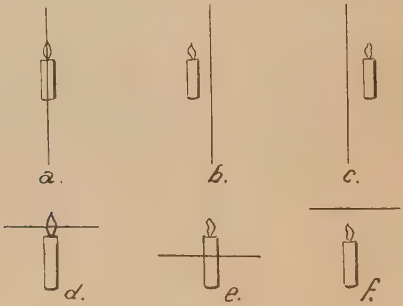
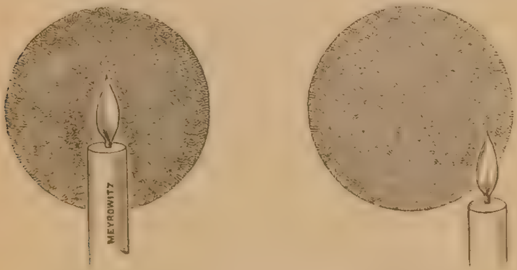


FIG. 5.—Maddox rod before right eye.
a. Lateral balance; *b.* Esophoria; *c.* Exophoria; *d.* Vertical balance; *e.* Right hyperphoria; *f.* Left hyperphoria.

siderable distance from the axial ray are refracted away from the fovea; and should an ex-central portion of the lens be brought opposite to the pupil, the image of the light will not fall on the fovea. Hence, the method is open to the objection of the prism test. But when carefully prepared and properly used, the test has every advantage of the Maddox rod and is superior to the prism test.

4. The Parallax Test.—The patient is asked to gaze (as in the other tests) at a light 20 feet distant on a dark background, and each eye is covered alternately, thus destroying binocular fixation. As the cover is carried from one eye to the other, the patient is asked if the light seen by the uncovered eye has jumped from the position occupied by it when previously seen by the other eye. Even slight movement will, after a few trials, be remarked by the patient, and he can accurately state that the light has moved, its direction and extent. For example: in heterophoria there will be a perceptible jump as the cover is shifted from one eye to the other. If the light moves in the same direction as the cover is carried, exophoria, and if in the reverse direction, esophoria is present. If the light shifts down when the right eye is uncovered, there is a right



Orthophoria. Heterophoria.

FIG. 6.—The convex spherical test.

hyperphoria, and if the reverse, left hyperphoria. The movement of the light may be controlled by prisms, and the degree of the prism which stops this apparent change of location of the light will be the measure of the heterophoria. This test is quite different from the objective cover test. By that method the observer determines *without the coöperation of the patient* whether an eye, while covered, has been deflected from parallelism with the other when gazing at a distant object; and it is then known as

5. **The Cover Test.**—The patient is requested, while standing in a good light, to gaze at a small object 20 feet or more distant. The eyes are screened alternately. By watching the eye the instant the cover is removed, no movement will be seen in orthophoria because the visual axes are parallel, but an excursion will be noticed in heterophoria. As the cover is carried to and fro from one to the other eye, if marked esophoria be present, the eye uncovered will move in a direction exactly contrary to the movement of the cover; *e. g.*, if the cover is carried from the left to the right eye, the left eye will jump to the left, and *vice versa;* and the prism, base *out*, before either eye that stops this jump or movement will be the approximate measure of the heterophoria. The method is applicable to all high-grade heterophorias of whatsoever kind.

6. **The Cobalt Test.**—A glass stained with cobalt oxid and ground to fit the trial frame has the power of intercepting all the rays of the spectrum excepting the blue and the red. The image seen by an eye before which the cobalt glass is placed will be much smaller than the real image, and will have a red center with the blue halo, or a blue center with the red halo; the former in myopia, the latter in hypermetropia and emmetropia. This test has the advantage over all the others in that it does not refract the rays before they enter the eye, and in all cases the rays

pass through parallel and are focused on the fovea. Hence, f deviation be discovered it must be the result of muscular nsufficiency and not due to inadequacy of the method of diagnosis. The method is valuable only in cases of 2° or more of vertical and 4° or more of horizontal imbalance. Fusion power is largely destroyed, and in the presence of any considerable heterophoria the patient will see two images, one the clear light, the other the smaller blue-red flame. In orthophoria, both images fall upon the fovea and the composite light will be the clear light tinged with red and blue. In heterophoria, the cobalt image will be

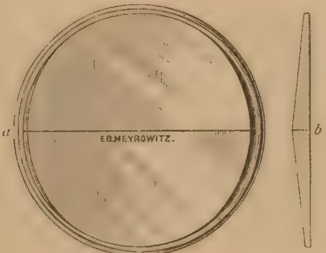


FIG. 7.—Maddox double prism.
a. Front view.
b. Sectional view.

separated from the clear image according to the kind and degree of the defect. In esophoria, for example, it will be on the side of the eye covered by the cobalt glass and on a level with the true light, the interval between them depending, as in other instances, upon the degree of the defect.

7. **The Maddox Double Prism** consists of two 4 prisms, bases together, fitted into an ordinary test glass cell and held before one eye. (Fig. 7.) It will cause the light as seen by that eye to be double (monocular diplopia). If now the other eye be uncovered, a third, which is the true image, will

appear midway between the two and directly on a line with them (orthophoria). (Fig. 8.) 1. Let the prism be so held that two images are in the vertical meridian (basal line horizontal). The true, or middle image, will be out of line, to the right or left, according as there is exophoria or esophoria. 2. Let the two images be on a horizontal line (basal line vertical) when the true image will be above or below this line in hyperphoria. The test is facilitated when the double prism is ground in red instead of clear glass. Another application can be made of this prism by observing whether the middle or true image is equidistant

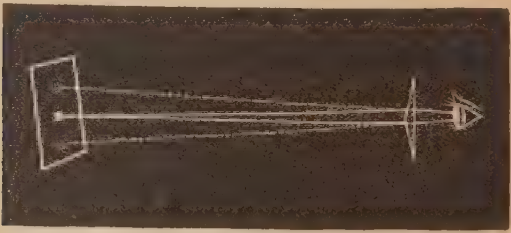


FIG. 8.—Effect of a double prism on a beam of light. (Maddox.)

from the two false (red) ones. If not, the prism necessary to restore it to the orthophoric middle position will be the measure of both the kind and degree of the heterophoria This prism is usually applied to investigating the tension of the oblique muscles. If they are faulty in action, th middle line is tilted up or down at the right or left. (Se chapter on Cyclophoria.)

TESTS FOR READING DISTANCE.

The Dot and Line Test.—The principle is exactly tha described for the prism test at 20 feet. It was devised b v. Graefe for testing insufficiency of the interni only.

prism of 8° is held base up, or down, before one eye. The patient gazes at a small black dot, through which a line is drawn, on a card held at the ordinary reading distance. If the prism is placed base down before the right eye, the two dots will be seen on the same line in orthophoria. In lateral insufficiency both dot and line will be doubled, the upper belonging to the right eye. The false image will be to the right (esophoria) or to the left (exophoria), according to the lateral deviation. In hyperphoria, with the test-card and the prism changed from the vertical into the horizontal position, the false dot and line will be above or below the true dot and line. In balance of the vertical muscles the false and true dots will be on the same vertical line. In our judgment, the dot alone forms a superior test, since the line maintains, in part, the desire for fusion and serves, in a measure, to prevent independent movement of either eye. A small printed word may be similarly employed. The Maddox test for the near, based also on prism displacement, provides a scale for the measurement of the angular deviation without other means. (Fig. 9.)

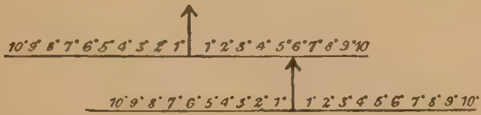


FIG. 9.—Maddox test for the near point.

COMPLEMENTARY TESTS.

In making up our minds as to the advisability of either optical or surgical treatment of heterophoria, the relations of the defective muscles and their synergists to the antagonistic muscles must be given due weight. For instance, it is not enough to say a patient has 3° of exophoria to induce treatment for exophoria, but this diagnosis should be confirmed by showing a corresponding change from the normal relations of abduction and adduction, that is: a diminished prism convergence and an increased prism divergence. In hyperphoria, the diagnosis should be substantiated by an increase of the supraduction or corresponding decrease of the infraduction. Thus, right hyperphoria of 2° would indicate that right supraduction was stronger by that amount than right infraduction.

In all examinations to determine muscular relations and anomalies, vision should be as nearly normal as practicable, and when an error of refraction exists, the correction should be worn at the time of making the examination since, in the great majority of cases, the refractive trouble influences muscular relations, as has been explained.

MUSCULO-DYNAMICS.

No tests for muscular coördination are complete that do not take account of the relation to each other of the various conjugate rotations of the globes, the practical determination of which is based upon the ability of the muscles to overcome prisms base up, down, in, or out, while both eyes are fixed on a small object at 6 m. or at the ordinary reading distance. A prism differs from a piece of ordinary plane glass only in the fact that its two sides (or refracting surfaces) are inclined toward each other forming an edge or angle. By reason of this inclination of the sides of a prism, a beam of light, instead of passing through it without change of general direction (as in the case of window glass), is bent from its course toward the broad end or base of the prism. (See Fig. 10, p. 71.)

If the eye be placed at *B* in the path of the beam after it emerges from the prism, it will not see the candle at *A*, its true position, but in the direction of the line *B C* projected through the prism along the dotted line to *A′*. Hence, there is displacement of the object in the direction of the angle of the prism, and on this phenomenon is based the law that prisms displace objects in the direction of their edge, angle, or apex. If, when the eyes are directed toward an object 20′ or more distant, a 6° prism be placed, base in, in front of the right eye, it so displaces the object that the image falls to the inner side of the fovea, and unless the eye by rotation temporalwards effects a corresponding inward displacement of the fovea centralis, diplopia is inevitable. The above elementary principle may be illustrated in its application to the prism test for determining heterophoria or orthophoria.

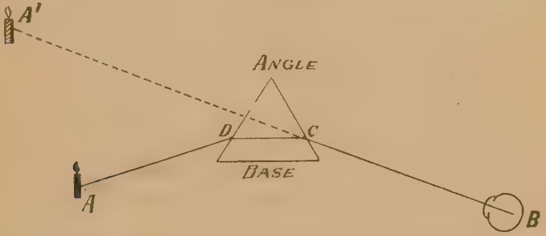
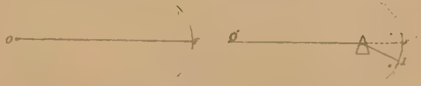


FIG. 10.—Dotted line indicates direction which the projected beam takes.



Right retina (R). Left retina (L).

FIG. 11.

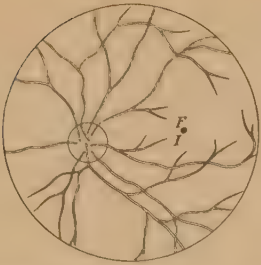
In Fig. 11, *O* is the object, *O F* the ray proceeding from the object to the fovea, *F*, of the right eye (*R*), and *O′ F′* the ray from the same object which if not interrupted by the prism would fall upon *F′* in the left eye, but a prism placed base *down* before that eye refracts the ray to a portion of the retina beneath the fovea. Since the lower half of the retina refers its impressions to the upper part of the field, *O'* will be projected above *O* and in a vertical line with it in orthophoria, or to one or the other side of *O* in lateral heterophoria. To test for hyperphoria or hypophoria the refracting prism is placed with its base in. The image of *O* will then be refracted to the nasal side of the fovea of that eye before which the prism is placed, and if hyperphoria exists one image will be seen higher than the other.

According to the law of projection, if an image falls to the right of the fovea the object *O* will be projected to the left portion of the field; if to the left, to the right portion of the field; if below, to the upper; and if above, to the lower portion of the field In Plate I, *A* is the fundus of an eye before which no prism is placed; *B*, fundus before which prism is placed base *down*, *I* representing the new position of the image in the lower half of the retina; *C*, prism base *up*, *I* displaced to the upper half of the retina; *D*, prism base *in*, displacing *I* to nasal half of the retina; and *E*, prism base *out*, displacing *I* to temporal half of the retina.

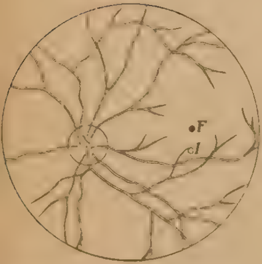
If the prism is not strong enough to overcome the power of fusion (in other words, to destroy binocular vision), the eye before which it is placed will be rotated so that the fovea will be moved from its original position to that occupied by the image. This faculty of setting aside by rotation the transient diplopia thus induced is known as the ability to overcome prisms, or the prism-rotation.

The student must not confuse prism-rotation in the above sense with the ordinary rotation of the eye; for instance, the abductors will normally overcome but 8° to 10° of prism base *in* (external prism-rotation), whereas the eyes can be rotated temporalwards from 48° to 53° from the primary position.

FIG. A

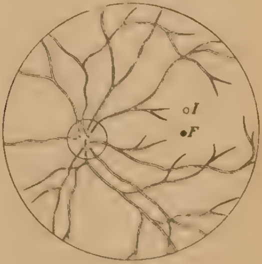


F = fovea. *I* = image. No prism. *I* falls on *F*.

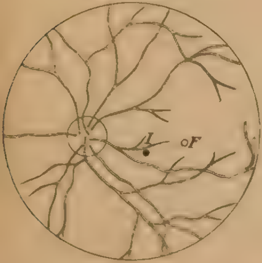


Prism base *down*. *I* falls below *F*.

FIG. C

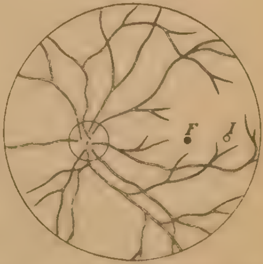


Prism base *up*. *I* falls above *F*.



ism base *in*. *I* falls to nasal side of *F*.

FIG. E



Prism base *out*. *I* falls to temporal side of *F*.

PLATE I.

When the prism is first interposed the right eye sees the image at *A'* (Fig. 12), but in response to the fusion impulse it is rotated outward so that the fovea is moved from *F* to *F'*, where the image may fall directly upon it and binocular vision be thus restored.

The limits to the power of rotation of the eyeballs are fairly well defined in the various meridians as measured by the prisms that they overcome. Beyond these limits fusion is no longer possible and diplopia ensues, hence the following terms:

FIG. 12.

Abduction or prism divergence.

Adduction or prism convergence.

Supraduction or vertical divergence as measured by prisms placed base *down* or angle *up* before either eye.

Infraduction or vertical divergence as measured by prisms placed base *up* or angle *down* before either eye.

To test abduction the prisms are placed base *in* before either or both eyes.

To test adduction the prisms are placed base *out* before either or both eyes.

[illegible]

To test supraduction the prisms are placed base *down.*

To test infraduction the prisms are placed base *up.*

Formerly prisms were made square, but they are now so shaped that they may be fitted into the cell of an ordinary trial frame.

It is the general custom to have the patient seated about 6 m. away from a small electric light or lighted candle, directing him to fix his gaze on the light. Prisms are then interposed with their bases up, down, in or out respectively, until permanent diplopia is produced. The highest prism that can be overcome, or with which single vision is still possible, is the measure of rotation of the eyes in the direction of the apex or angle of the prism: *e. g.*, if the eyes overcome an 8° prism, base *in*, but not a 9°, adduction = 8°; if a 15° prism, base *out*, but not a 16°, adduction = 15°; if a 2° prism, base *up* or *down* before the right eye, but not a 2½°, right supraduction or infraduction respectively of 2° is shown. It should be borne in mind that a prism with its base down before one eye is equivalent in its action to the same degree of prism with its base up before the other eye.

Various devices are employed by different workers for the estimation of the rotational powers of the ocular muscles, principal among which are single prisms, the prism battery, Risley's rotary prism, Jackson's rotary prism, Stevens's phorometer, Wilson's phorometer, Stevens's tropometer and clinometer, and Landolt's ophthalmo-dynamometer. The method just described, upon the principle of which most of the others depend, is that by the use of the single prisms. A more convenient method of interposing one prism after another in testing the ocular rotations is by the prism-battery, first suggested by Noyes and later modified and warmly advocated by Gould. (Fig. 13.)

The series begins with one-degree prisms on each side, and increases by integers of 1° to 20°; thus by steps of 2° each, all degrees of abduction and adduction from 2° to 40° may be measured. "Each battery is revolved at pleasure, being fixed by a pivot mechanism above and below, and temporarily held in the position desired, by clutches at the sides. To measure adduction each battery is placed bases *out;* to measure abduction, each battery is revolved so that the bases are in. The open central space permits the lenses to be brought close to the eyes, without interference with the patient's nose." This instrument is applied only to the lateral rotations. For measuring the vertical rotations, we would suggest a similar battery (single) carrying weak prisms (from ¼° to 5°) bases up or down. By reversing the battery both supra- and infraduction could thus be readily and accurately determined. However, all these requirements and necessities are beautifully met in what is known as the "prism mobile," on the principle first suggested by Sir John Herschel, who showed how, by placing two prisms in apposition and rotating them in opposite directions, we can produce the effect of a single increasing prism. By far the neatest application of Herschel's idea is that of the Rotary Prism

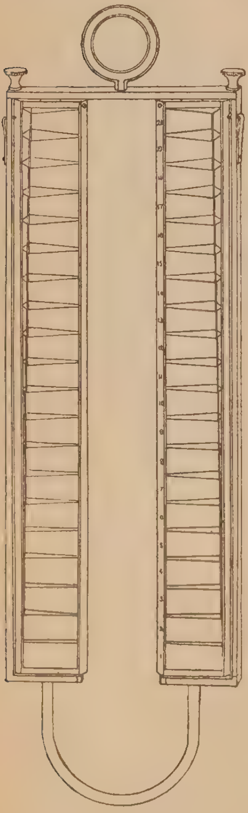


FIG. 13.—Gould's prism battery.

designed by Risley (Fig. 14), to be fitted into an ordinary trial frame. On turning the little mill-edged screw, the two prisms of 15° each rotate in opposite directions, the strength of the resultant prism showing on a graduated scale engraved on the blackened front of the containing cell. However, by occasioning chromatic aberration and prismatic astigmatism, all rotary prisms share the disadvantage of lowering the visual acuity in one eye only, and thus the desire for single vision is lessened more than if the prism effect were divided between both eyes. Maddox has therefore suggested an apparatus in which one prism revolves before each eye. It is practically the same as the prism arrangement in Stevens's phorometer, and is useful only in measuring lateral rotations. A similar instrument for measuring vertical rotations could easily be devised by having the prisms fitted into Maddox' device, bases *in*, and then rotating them in opposite directions. To produce more delicate results in the lower prismatic powers, Jackson employs in his rotary prism three prisms, one 15° stationary prism and two rotating ones of 7½° each.

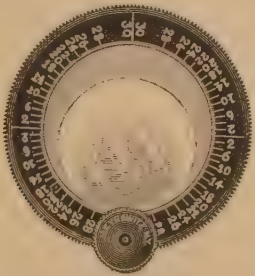


FIG. 14.—Risley's rotary prism.

A radical improvement for some purposes has been made over the Herschel combination in Stevens's phorometer (Fig. 3, p. 61), which divides the effect between the two eyes. It has a standard and an adjustable arm furnished with a spirit level, and a slide containing two cells, in each of which rotates a disc carrying a prism of 4°. Each disc, furnished with cogs, can communicate movement to the other by a small gear cogwheel placed between the discs.

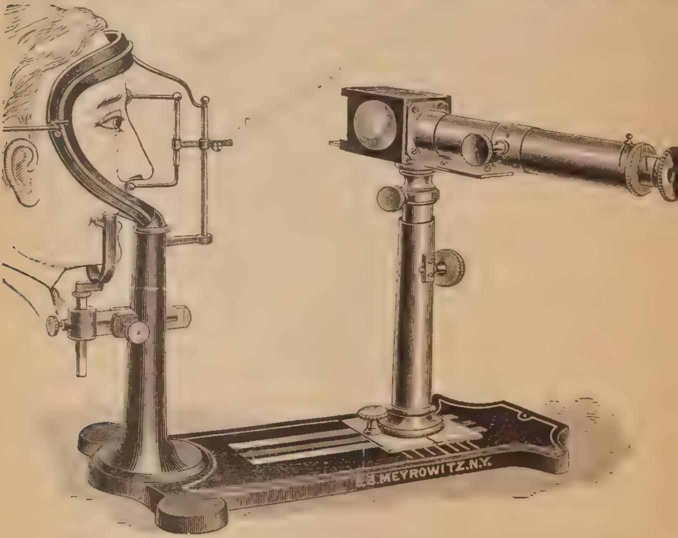


FIG. 15.—The tropometer (*Annales d'Oculistique.* English edition, July, 1895).

While the instrument was primarily devised to measure deviation-tendencies of the ocular axes (heterophoria), it can be applied with great exactness to estimation of the ocular rotations, especially the vertical ones. The same may be said of Wilson's phorometer. The tropometer of Stevens (Fig. 15), by means of which we learn the comparative rotations of the eyes, often enables the surgeon to accurately localize a defect of rotation in some particular meridian. "It consists essentially of a telescope, in which an aerial image of the cornea is formed near the eye-piece. A scale, graduated to measure the rotations of the eye in all directions, is placed at the point where the image is formed." After placing the head in the head-rest, so that the glabella and the commissure of the upper lip are exactly vertical, the patient fixes on the center of the object-glass. Under the direction of the surgeon, the eye

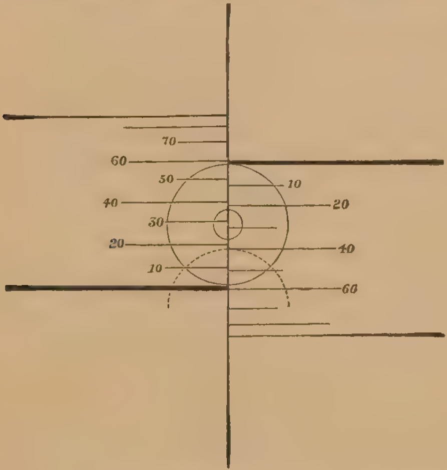


FIG. 16.—Scale for tropometer.

under examination is rotated to the fullest extent in the four principal meridians while the head is kept absolutely immovable. The effect registered on the scale by movement of the cornea is shown in Fig. 16. These rotations are, approximately:

Upward	33° (of arc)
Downward	50° "
Inward	48° to 53° (of arc)
Outward	48° to 53° "

and they represent the four principal secondary positions. In all the degrees of the circle between them, the rotations will be accomplished by the combined action of the muscles normally turning the cornea to those positions, and when these points have been found and connected they map out the *monocular field of fixation*, which varies much, however, according to different observers. The average is probably as follows:

Up.	Up and out	Out.	Down and out.	Down.	Down and in.
40°–50°	35°–50°	35°–55°	35°–60°	40°–70°	25 60

In.	In and up.
40°–48°	40°–55°

The discrepancies arise from variations in the normal power of the muscles of one individual as compared with another, and the amount of attention and effort of which an individual is capable. The *field of binocular fixation* comprises that part of the visual or form fields that is visible to both eyes at the same time without movement of the head, but with movement of the eyes. It is not necessarily identical with the field of binocular single vision, although the limits are practically the same. Near the periphery of the field of binocular fixation, the images of the object may not fall upon the yellow spot of each eye, but upon adjoining portions of the retina, when insuperable diplopia will be manifest. The field of binocular vision is smaller than the united fields of each eye, and that of binocular single vision is still smaller. The limits of both may be readily determined by perimetric measurement, using one or two small white dots on a black background, or black dots on a white background. Those points on the periphery where the single dot doubles or blurs, represent the limits of the field of single vision. They are approximately:

Up	60° (Pooley)	45° (Duane.)
Down	70° "	70° "
Right and left,	60° "	55° "

In old age the rotations are lessened, and in determining them at any age, the form and position of the globe in the orbit must be taken into consideration. Such wide variations as are shown in the averages given above, are confusing to one who endeavors to determine whether a certain movement is normal or otherwise. An excess of 33° upward or of 50° downward rotation should direct suspicion to the vertical muscles; and if, in the presence of esophoria or exophoria, the temporal and nasal rotations are normal, operation on the lateral muscles will scarcely be justifiable and then only when the vertical muscles have been exonerated of causative influence by careful estimation of their rotations; this is especially important because high-grade lateral deviation-tendencies are often an indication of unequal vertical tensions. It may be well to note, in passing, that the surgeon may learn by means of the tropometer the peculiarity of his own muscular status with respect to the horizon before proceeding to the use of the clinometer (Fig. 17, p. 82), also devised by Stevens, for accurate study of the horopter in all cases of anaphoria or anatropia; also cataphoria or catatropia. In case the observer is exophoric or esophoric, the tubes may be adjusted in divergence or convergence, and in case of hyperphoria a correcting prism may be placed in a clip next the eye.[1]

PRISM MEASURE OF ASSOCIATED MOVEMENTS.

It is customary to express the power of rotation of the eyes in the vertical and horizontal meridians in terms of the strongest prism, base up, down, in, or out, with which single vision is still possible. Of the four principal rotations, three of them, namely, abduction, supraduction, and infraduction, are fairly constant in quantity, and authorities agree more or less in fixing the power of

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[1] Monocular diplopia is the result of irregular corneal or lenticular curvature, or of disease of the foveal region, and bears no relation to the subject under discussion.

Abduction	at from 6° to 8°
Supraduction	at from 2° to 3°
Infraduction	at from 2° to 3°[2]

Much depends on the method used in the determination. The above figures are those furnished by single or separate prisms held before the eye one after another. Rotary prisms, especially as employed by Maddox, or any

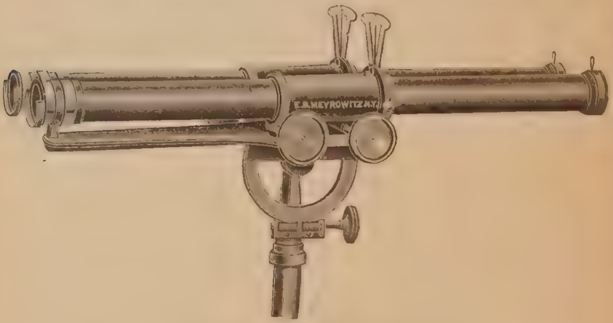

FIG. 17.—Stevens's clinometer.

similar apparatus of graduated increment, indicate higher average powers of rotation, viz.,

Abduction	8° to 10°
Supraduction	3° to 4°
Infraduction	3° to 4°

The fact seems to stand that if, at the first trial of abducion, it is found to equal 7°, there will be little if any variaion in its degree, no matter how often it is tried on the same day or succeeding days, provided only that the same method is used each time. The same relative constancy is found in supraduction and infraduction. Not so, however, with adduction, which by its variation from day to day, and even from hour to hour in some cases, has thrown much confusion into the subject of muscular anomalies. Perhaps this confusion hinges on the neglect of many observers to distinguish between primary adduction (or the prism convergence manifested at the first trial) and cramped or trained adduction (the prism-convergence possible when the patient has learned to temporarily dissociate accommodation and convergence). Moreover, personal equation counts for no little variation in the results of different investigators. Gould works by estimating first the *upper diplopsic point*, corresponding to that degree of combined prism strengths at which definite diplopia appears; proceeding downwards from this point again towards zero, he finds the lower diplopsic point, or that degree of combined prism strengths at which binocular single vision is again resumed (in other words, at which fusion again asserts itself), and then estimates primary adduction as the point midway between these two other points. Stevens's latest utterance is to the effect[3] "that although an exact standard of adduction is not to be expected, should the adducting ability fail to reach 50° (prism) after a reasonable amount of practice, it is likely to be deficient." Risley[4] found the average adduction for 20 feet in 25 non-asthenopic individuals to be 25°, while Banister,[5] who conducted a series of careful studies on 100 soldiers all in fine physical condition, found the average adduction for 20 feet to be 14°.

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[2] These figures hold for 20 feet only. For any less distance abduction would be greater and adduction less; for instance, at 15 feet abduction equals about 10°, and if the eyes are steadily approached, a point will finally be reached where abduction and adduction are equal.

Norris and Oliver, System of Diseases of the Eye, Vol. ii, 1898.

[4] *Univ. Med. Mag.*, January, 1895.

[5] *Annals of Ophthal.*, January, 1898.

It seems to the authors that Banister's statistics indicate plainly the average degree of *primary* adduction, while Stevens and Risley's figures have reference more to *trained* adduction. However, it must be borne in mind that convergence and divergence are acts so intimately bound up that it is almost impossible to dissociate them, so that the study of one side of the phenomenon necessarily involves the other. For instance, in overcoming prisms bases in (or fusing lights through prisms held bases in before the eyes), a stimulus to divergence is created. The eye before which the prism is placed is compelled to diverge in order that a single image of the light may be preserved. It will be noticed that we speak here of divergence and not of the external rectus, because all muscles which help to turn the eye out are primarily included in the test. When the prism is placed before one eye only, all of the diverging is done by that eye, and the other unwaveringly maintains its direction toward the object focused. Yet it does not follow that the testing is confined to the eye that turns, for the turning eye is held in its divergent position and the other in its straight position by a contraction of all the external muscles, and therefore we are, in a certain sense, testing convergence and divergence at the same time. The completed action is really a very complicated one. For example: a 6° prism is held base in before the R. eye; in order to overcome the momentary diplopia induced by the prism, the abductors of the R. eye are called into play and that eye diverged, as can be seen through the prism. Strangely enough, the L. eye is held straight, notwithstanding the general rule that movement of one eye in any direction is always accompanied by a similar associated movement of the other eye[6] and that therefore the adductors of the L. eye tend to turn that eye in the same direction as its fellow. To neutralize this latter impulse the abductors of the L. eye are called upon, and their action necessitates, in turn, action on the part of the adductors of the R. eye, which would immediately bring the R. eye back to parallelism were not the abductors of that eye fully occupied in keeping it in such position as to avoid diplopia. Hence the introduction of the 6 prism disturbed divergence primarily and convergence secondarily. The same phenomenon occurs when prisms are placed bases *out* before one or both eyes. In testing supra- or infraduction a similarly complicated act is excited. Thus: when a 2 prism is placed base down before the R. eye, the image falls on the R. retina below the fovea, in consequence of which the elevators contract and the R. eye rolls up, turning its fovea until it reaches the position of the displaced image. Naturally an equal stimulation is sent to the elevators of the L. eye (according to the above-mentioned rule for associated eye-movements), but a change in the position of the L. fovea would destroy the test so that the upward impulse of the L. eye must be met by an equal downward impulse, and this in turn stimulates a downward impulse of the R. eye, which is prohibitive because of the diplopia that would be sure to follow. Hence, while the elevators and depressors are in equilibrium, they are only artificially and temporarily so. We are in a sense determining the power of elevation as compared with that of depression, but we have been really investigating the limits of equilibrium by prism stimulation of allied and of antagonistic muscles.

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[6] Movements induced by prisms not too strong to permit fusion constitute the sole apparent exception to this rule.

Further knowledge of the relations of convergence and divergence may be gained from the use of Landolt's ophthalmo-dynamometer (Fig. 18). Just as in the study of the accommodation, we learn its amplitude by estimating the position of the punctum proximum and the punctum remotum, so in the study of convergence we find that its amplitude is equal to the difference between the maximum and minimum of convergence. Landolt's device consists of a blackened metallic cylinder which is to be

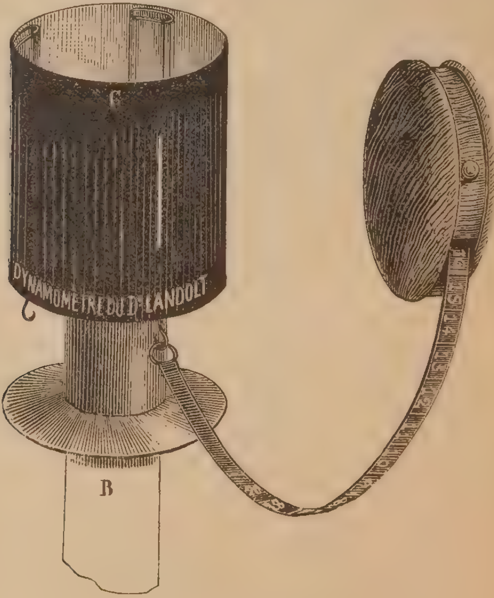

FIG. 18.—Landolt's ophthalmo-dynamometer.

fitted on to a candle. The side of the cylinder turne toward the eye presents a narrow slit (0.3 mm.), which is illuminated by the flame of the candle and serves as fixation-object. Beneath is attached one end of a spring tape-measure, graduated on one side in centimeters and o the other in the corresponding numbers of meter-angles (or, what amounts to the same thing, in diopters). To find the punctum proximum of convergence, the case holding the tape-measure is held beside one of the eyes and the cylinder drawn about two-thirds of a meter away from it, directly in front of both eyes, with the illuminated slit turned toward the patient, who then fixes on the light streak while it is moved directly in the median line closer and closer to the eyes. The moment the streak of light begins to broaden or double, the point of greatest possible accurate convergence has been reached, when one side of the tape will indicate in centimeters the distance of the punctum proximum of convergence, and the other side the corresponding maximum of convergence in meter-angles; for instance, 11 centimeters correspond to 9 meter-angles. In the mentally dull, the examination can be made much easier by having a colored glass before one eye so that doubling of the light streak will be recognized the instant it occurs.

The minimum of convergence is determined by the following method: Make the patient fix the flame of a candle at a distance of six meters or more and find the strongest prism *base in* before one eye that can be overcome without causing homonymous diplopia; then divide the number of the resultant prism by 7, to obtain *approximately* in meter-angles the amount of deviation of each eye. Thus, if abduction at 20 feet is 7° the minimum of convergence is 1.00 meter-angle; if 8° it is 1.14 meter-angles; and if 6° it is 0.85 meter-angle.

In the normal state the average maximum of convergence, (p) is about 9.5 meter-angles; and the average minimum of convergence (r), about -1 meter angle; therefore the mean amplitude of convergence (a), as expressed by Landolt,[7] would be,

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[7] The Refraction and Accommodation of the Eye, 1886.

$$a = 9.5 - (-1) = 10.5 \text{ meter-angles};$$

or if the maximum be 12.5 meter-angles and the minimum 1.5 meter-angles the amplitude of convergence would be 14 meter-angles. The calculation is one of simple addition and subtraction, and it will be observed that it takes account not only of convergence, but that it also reckons the extreme limit of divergence (as found with prisms) as the measure of the *minimum of convergence*, a fact that the student will do well to ponder and settle for himself before proceeding to the study of any practical office problems in anomalies of convergence and divergence. As above stated, the two acts are so intimately bound up that it is relatively impossible to dissociate them, and investigation of one side of the question necessarily involves the other.

Careful weighing of all the facts connected with these phenomena seems to warrant the following tentative conclusions:

1. That the degree of adduction (prism-convergence), for 6 meters given by most writers as proper in the first office-examination cannot be reached by healthy eyes except after practice in prism-convergence.

2. That primary adduction at the first office-examination will often not exceed 20°.

3. That adduction (prism-convergence) can often be quickly trained to 50° and not infrequently to 75°, and in some cases even to 90° or 100°.

4. That estimation of the *amplitude of convergence*, after the method of Landolt, is the best test of the real power of convergence, and its limits.

5. That abduction as found at the first office-examination is fairly constant, and in orthophoria will rarely fall below 7°.

[illegible]

That the ratio between abduction (prism-convergence), and abduction (prism-convergence), for 20 feet, ranges

[illegible]

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Second line from bottom
Read "adduction" instead of "abduction."

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Read "prism-divergence" instead of "prism-convergence."

ed as
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largely a matter of personal equation.

Section III.—ESOPHORIA.

Symptoms.—The symptoms of this tendency of the visual axes toward each other are local or ocular, and general or reflex. They vary according to the degree of the esophoria and the susceptibility of the patient. The local symptoms are in no wise distinctive. They are: pain in the eyes and in their immediate neighborhood; flushing of the conjunctiva after use; photophobia; lacrimation; blurring of the print in reading; and inability to continue for any length of time the effort of looking at near or distant objects without becoming drowsy. As will be seen, the same symptoms may depend upon hyperopia or astigmatism, or any other muscular or refractive error. The reflex symptoms are exceedingly complex and unstable, and they vary from an occasional slight headache to intense migraine; from a simple restlessness to serious functional nerve-diseases; from a chronic insignificant disturbance of digestion to loss of appetite, interrupted digestion, and vomiting. A peculiarity of esophoria, not found among symptoms of refractive or other muscular anomalies, is that the patient often complains of seeing his nose, especially in close work. This symptom is largely imaginary, for the patient is no more inclined by esophoria to turn his eye in so far that coordination will be lost, than he is to have double vision for all objects. The complaints are not limited to the time of using the eyes, but often follow for many hours after a continued or unusual strain. Identical symptoms, although perhaps not so severe, arise from gazing intently in the effort to see clearly a distant object. Esophorics invariably are disturbed by traveling in the train or by looking at rapidly moving objects. The symptoms are due, not directly to the internal muscles, but to the constant effort under which the patient labors to prevent abnormal convergence. A tendency inward can be controlled only by an equivalent tension on the muscles of divergence. If the defect is so high that the external muscles cannot constantly maintain coordination of the visual axes, and one eye turns inward, the symptoms may be immediately relieved, but the patient will complain of diplopia. It is not always the highest degree of defect that gives rise to the severest symptoms. The disturbances above described never arise, for instance, in cases of manifest internal squint. It can be readily understood that the constant strain on the abductors to maintain parallelism of the visual axes may give rise, in certain subjects, to alarming reflex neuroses. No set of muscles, in any part of the body, can be in a state of constant contraction without making a deep impression upon the nervous system. Indeed, hypnotic states are induced through an analogous process. The severity, then, of the symptoms must depend almost entirely upon the susceptibility of the individual to abnormal impressions, and the patients who suffer the most in esophoria are those who habitually complain severely from apparently insignificant causes.

Etiology.—The causes that contribute to the abnormal tendency of the visual axes inward are local and constitutional. In many cases, it is quite impossible to satisfactorily explain the origin or the existence of esophoria. It may be stated, however, in general terms, that it is found most frequently in persons of a neurotic disposition who have low grades of H. or H. + As. The susceptibility of the nervous system plays a most important rôle in the causation of muscular anomalies, particularly esophoria. By that expression is meant an abnormally acute impressionability of the nervous system induced by lack of proper nourishment from deficiency of oxygenated blood, a stasis of the venous or arterial supply from disturbance of the circulatory system, in short, a neurotic disposition. That an underlying predisposition to irregular nerve-activity is an essential contributing factor in the development of the muscular imbalance and to the symptoms accruing from it, is demonstrated by the well-known fact that many individuals, who have the theoretic local conditions assumed to give rise to muscular asthenopia, use their eyes constantly without discomfort ; furthermore, in the absence of the local causes that are conceded to produce esophoria, and in the presence of those that are usually causative of exophoria, esophoria is often present and gives rise to no symptoms. Identical inconsistencies are illustrated in the study of the etiology of other than eye-diseases. Two persons exposed to the same influences will be seized with different affections or one will entirely escape. It is not strange, therefore, that we are often at a loss to ascribe the mysterious unbalancing of the ocular muscles to a competent cause, and are compelled to invoke the aid of the supposition of a deranged nervous organization.

The interdependent, constant, tenacious relation between the intra- and extra-ocular muscles furnishes an explanation of the existence of esophoria in a majority of cases. In health there is a relative range of accommodation and convergence; every contraction of the ciliary muscle is accompanied by an equal contraction of the adductors, and, conversely, stimulus to the accommodation means equal stimulus to convergence. In emmetropia the degree of the meter-angle convergence is determined by the diopters of accommodation. Thus, accommodation of 3 D. induces convergence of one-third meter-angle. Now, if for any reason an abnormal amount of accommodation is required to read at the ordinary near point (thus overstepping the normal range), a correspondingly increased demand on convergence is made, and a tendency to converge the visual axes to a point within the desired reading distance results. When, notwithstanding this unbalanced relation, the normal convergence of the visual axes is maintained, it can be only at the expense of the relative accommodation and convergence, and in any case it entails an unusual resistance on the part of divergence. If the range of the relative accommodation and convergence is not exceeded in this effort to secure binocular fixation at the reading distance, there will be no esophoria and no symptoms, and this probably explains the exemption of certain individuals. The three refractive conditions that most frequently give rise to a disruption of the relative range are hyperopia, hyperopic astigmatism, and low myopic astigmatism. To overcome hyperopic states of the refraction and thus sesure good acuity of vision for both distance and near, unusual demand is made on the ciliary muscles, which in turn stimulates all the other muscles supplied by the 3d nerve. Hence, an inward tendency of the visual axes is originated, but this, in the interest of binocular single vision, is suppressed and the condition remains a tendency only. The same reasoning applies to hyperopic astigmatism ; myopic astigmatism of low grade is also a frequent and potent cause of ciliary spasm, and thus may be classed among the causes of esophoria.

Treatment.—When, by means of one or all the tests described on page 59, inward tendency of the visual axes has been repeatedly and conclusively determined, we are confronted with the practical problem of relief. The method selected depends upon (1) the severity of the symptoms, and (2) the degree of the esophoria as measured by prisms, bases out. We recommend the adoption of the following course:

First.—In all cases the estimation of any optical defect under complete paralysis of accommodation and the wearing, for some weeks, of as nearly a full correction as can be borne with comfort. This is essential. No other treatment directed to the restoration of the lost equilibrium of the muscles should be inaugurated until glasses correcting the ametropia have been worn sufficiently long to remove any pernicious influence born of constantly over-taxed accommodation. The experience of all ophthalmologists confirms this statement, and no surgeon of judgment or ability will apply treatment directly to the muscles until he has given the patient the opportunity to cure himself by wearing glasses. Any coexisting general disturbance must also be relieved by appropriate medication. Diet should be regulated; exercise in the open air instituted; the time devoted to reading restricted; in short, every means tending to diminution of the unwonted excitability of the nervous system, or to derangement of any of the vital functions, must be employed.

Second.—Prism exercise and nerve sedatives. By means of frequent and interrupted use of prisms, we endeavor to strengthen the power and increase the range of divergence, and unless there is an actual deficiency of divergence, or an actual preponderance of convergence, many cases of recent standing may be effectually treated by this method. Abduction normally equals 7° or 8° when tested with a candle-light at 20 feet. In esophoria, abduction frequently falls to 1°, 2°, or 3°, and the *normal* ratio between abduction and adduction (about 1 to 3 or 4) is lost. Special apparatus to hold prisms have been devised, but beyond their convenience they offer no particular advantage over the separate square prism held by the patient. A prism of 6°, axis horizontal, is held *base in* before the patient's eye, and the patient is instructed to look at the candle-flame 20 feet distant. If his abduction equals only 3°, the prism will produce insuperable homonymous diplopia; but if he advance to within a few feet of the candle-flame, abduction increases as he advances, and he will reach a point where the two lights will fuse. He now recedes from the candle, maintaining its image single as long as possible. When the images have again separated he approaches the candle until the lights are fused, when he again recedes. This may be repeated a number of times, but no longer than five minutes at one sitting. On the second trial he will find that the lights are fused easier than at the first. At the third trial it will be still easier, and in the course of a few days he will be able to fuse the lights at 20 feet. This exercise should be continued until asthenopic symptoms have disappeared for some weeks. The prism may be held before either eye, or before one and then the other, and the effect will be the same. Fusion of the lights is not the result of a contraction of one or the other externus alone, but is accomplished by forced action of all the muscles which participate in divergence—an extremely complicated function. (See Weiland's article in *Arch. of Ophthal.*, January, 1898.) A useful adjunct to the prism exercise is the internal administration of the tincture of belladonna, hyoscyamus, or some of the other sedatives, in as large doses as can well be borne. The effect of these remedies is to diminish the spasm or abnormal contraction of the adductors and benumb the sensitiveness of the nervous system.

Third.—Correction by prisms. When, after appropriate trial by means of the above remedies, it is felt that something must be done, and operation is out of the question, the symptoms may be relieved by the use of prisms, bases out, which correct the larger part of the esophoria. The degree of the defect requiring the use of prisms

orrection [illegible]

varies from 3° to 10°. Defects under 3° may frequently be corrected without them, while those higher than 10° necessitate operation in nearly every case. The degree of the defect is determined in every instance by the result of testing at 20 feet. A constant relation does not exist between the convergence for distance and for the near point in cases where equilibrium has been lost. For instance, a defect of 7° or 8° for distance may be the same, or less, or more, at the near point, depending upon various concomitant conditions. The strength of prism for constant wear should be slightly less than the total amount of defect. It is a question whether wearing a prism correction does not develop an increase in the error; the very principle of its action forbids a cure. Under such treatment the tendency of esophoria is to advance, but whether this increase is due directly to the effect of wearing the prisms or to the unmasking of a latent defect is uncertain. Should the general health be improved by medication, change of environment, or occupation, etc., the defect may become less, but in no case can this improvement be ascribed to the prisms. It is oftener the case that stronger and stronger prisms will be required to relieve the symptoms until, finally, the case comes to operation. Our plan is to allow the patient to wear his prism correction without change as long as he is comfortable, allowing him to decide whether it shall be worn constantly or only for near work. In most instances, constant wearing only will give relief. It must be remembered that esophoric patients do not complain of headache or other symptoms consequent upon prolonged use of the eyes at the near point only, but intense gazing at objects in the distance, such as the preacher in his pulpit, looking steadily from a car window, or gazing at objects that are themselves moving, will give rise to these symptoms. The directions to the patient should be modified according to his occupation and his symptoms, allowing him to judge as to the occasions when it will be necessary to wear the glasses. Prisms in the position of rest give relief because, while worn, the patient's eyes are allowed to assume a position which is one of rest for them, and objects are held single by the refracting power of the prism—not by the strain on the antagonist muscles. In esophoria, the visual axes have become converged, and prisms, bases out, bend the rays proceeding from the object, outward towards the new position occupied by the foveæ centrales. Here the position of rest is not parallelism, but convergence. The tendency to turn has become an appreciable turning. Prisms must be regarded as crutches that will permit the patient to travel comfortably over his rough road, rather than as a means of cure. The strength of the prism ordered should always be less than the full defect. Otherwise, a tendency to develop whatever latent esophoria there may be, or to actually produce a higher grade, becomes manifest since the external muscles receive no stimulus to maintain equilibrium. If there be 6° of esophoria for distance, the prism ordered should be 4°, bases out, divided between the two eyes, that is, 2° for each eye; if 8°, 3° for each eye, bases out.

Fourth.—In all cases operation should be reserved as the final means of effecting cure; in low grades of esophoria, in fact in all grades of esophoria not bordering on esotropia, the means recommended under paragraphs 1, 2, and 3 should be given a proper trial and cannot be too much emphasized. Some authorities go even farther than this and state that no benefit whatever is derived from operation. Our experience does not justify this statement. We are convinced that many cases are operated upon that could be relieved by less radical measures, and, on the other hand, we have operated frequently where other means have been tried and failed, and have been entirely successful. The measure of success is the cessation of the symptoms and the restoration to equilibrium of the muscles. The condition known as equilibrium varies, as has been before noted, from esophoria of 2° for the near, to exophoria of 3° or 4° for the far. But it is our opinion that equilibrium or orthophoria is present only when, by testing with the Maddox rod, the streak of light passes directly through the center of the flame horizontally and vertically. Having decided that surgical procedure is necessary, we have the choice of advancement of the external recti or tenotomy of the interni. According to Landolt, tenotomy is never in place—advancement is the only surgical means. He bases this opinion upon the assumption that, in all cases, "the defect arises from a *loss* of divergence and not an *increase* or *spasm* of convergence; hence a weakening of the muscles which are not too strong is wrong principle." If esophoria depend on hyperopia or hyperopic astigmatism, in other words, if the abnormal convergence is a reflex from unusual activity of the accommodation, the tendency of the visual axes inward is not a result of an insufficiency of divergence, but a physiologic sequence to inordinate action of the ciliary muscle. Hence, in esophoria, tenotomy of the interni is more often productive of good results than advancement of the externi.

It is not practicable to state in precise terms the exact degree of esophoria that demands or justifies tenotomy, or to make an arbitrary law, any more than it is reasonable to say that in hypermetropia a certain number of diopters or fractions of a diopter must be thrown off from the full correction. The decision, as to operation, depends upon the effect of the methods of treatment previously outlined and on the severity of the symptoms. If we find that under prism exercise and internal medication the defect remains unchanged or slightly improves, and the symptoms become less and less annoying, even though the degree of esphoria be moderately high, operation is not to be recommended. And, on the other hand, if a moderately low degree is associated with severe symptoms and only partial relief results from the wearing of prisms, operation is advisable. In our own practice we are generally guided by the severity of the symptoms, and by the presence of diplopia with the cobalt glass. *After other means have been faithfully tried and failed*, esophoria of more than 10° will almost invariably demand surgical interference. In cases with less than this degree the symptoms may be controlled by the constant wearing of prisms. Prisms of more than 4° in each eye cannot, as a rule, be comfortably worn. Moreover, prisms are changeless and constant in their refraction, while the muscular conditions of the eyes are changing and inconstant. We are attempting to correct a live physiologic function by means of a dead, unalterable piece of glass. The degree of esophoria varies as above stated; in fact, different estimates may be reached by different observers at the same examination, therefore, allowance for this variance must be made in the prism correction. After operation and consequent reduction in the degree of esophoria, the conditions are more amenable to prism treatment.

Should the operation be limited to one eye or should both be included? We are dealing either with excessive convergence or defective divergence, and in either case we have to do with more than one muscle and more than one eye. Theoretically, therefore, the operation should be divided between the two eyes, unless we have reason to believe that the esophoria is the result of an organic muscular change, limited to one eye, or is due to a nebula of the

ornea, to high-grade optical defect, to vitreous or lenticular
pacity, or some other impairment of vision, indicating that
he esophoria proceeds from a local cause and is not the
esult of disturbance of innervation. Heterophoria is rarely
net with in cases where organic imperfections of one eye
ead to manifest squint and (usually to) amblyopia of high
legree, and may therefore be excluded from consideration
n this chapter. We are concerned with patients who suffer
rom no incurable affection of vision, have no organic lesion
hat can be determined, and who have, as a rule, fair acuity
f vision in both eyes. We cannot, therefore, admit that
he tendency of the visual lines inward is a monocular
ffection and that its treatment is consequently monocular.
n all cases both eyes should be made to share, as nearly
s possible, the deviation as measured by prisms. Where
louble operation does not seem to be indicated, the symp-
oms can be relieved by the other means suggested without
peration. It is a debatable point whether both eyes should
e operated on at the same sitting or the second operation
ollow several days after the first. The points in favor of the
ormer are, that under antisepsis the surgical procedure is
ree from danger; the incision is extremely small, involving
ut few tissues and these are superficial; the pain is insig-
ificant and confined to the dragging forward (either by the
ook or forceps) of the tendon and the cutting of its fibers;
emorrhage is unworthy of mention; the subsequent dis-
omfort is little if any more after the double operation; the
otation of each ball is similarly and cotemporaneously influ-
nced; the patient is gratified that the performance is com-
leted. The advantages of the single operation are: less
mmediately subsequent annoyance; the possibility that it
vill suffice for a cure. But such advantages are not worthy
f serious consideration when compared with those of the
lual operation. The progress of the operation must be

marked by measurement of effect according to the change in position of the images of the candle-flame at 20 feet, or of the streak if the Maddox rod is used. As often as necessary, the operation must be suspended and the result of the section of a few fibers determined, otherwise the operator is in the dark and his result is only approximate.

Section IV.—EXOPHORIA.

The conception of exophoria that seems to be nearest to physiologic truth is that the outward tendency of the visual axes results from a loss of convergence-impulse and hence must be considered as a passive rather than an active condition. With the exception of the comparatively few cases that may be described as anatomic (in which the fault lies in a structural deviation from the normal, in the insertion of the muscles of convergence or divergence, in the abnormally diverging orbits found in wide skulls, unusually broad muscles, or other anatomic peculiarities), the cause may be assigned in general terms to a loss of innervational impulse to the muscles of convergence. Hence, the muscles involved are precisely those affected in esophoria, namely: the interni, superior and inferior recti, and, according to Weiland, the inferior obliques. Instead, however, of undue contraction, resulting, as in esophoria, from over-stimulation of the accommodation, we have undue relaxation arising from a disturbed relation of accommodation and convergence, as in myopia. Moreover, exophoria is more often the result than it is the cause of a lowered tone of the general system, as in convalescence from low fevers, severe hemorrhages, confinement, and other exhausting affections. In esophoria the reflex symptoms may with moderate certainty be considered as emanating from the eyes, but in exophoria the eye-symptoms are primarily dependent upon the causes which have brought the system into a subnormal condition. These statements have an important bearing upon the treatment and should be carefully considered in deciding upon measures of relief. The most frequent and active cause is a myopic error of refraction. In myopia and myopic astigmatism of two or more diopters, the demand on the accommodation, when reading or otherwise using the eye at the usual near point, is less than in other states of refraction, since the lens, already too convex to bring parallel rays of light to a point, on the retina, is adapted for a distance inside of infinity, and the higher the myopia, the nearer to the eye is its far point; therefore the less need for contraction of the ciliary muscle. The muscles of convergence, deprived of the impulse associated with ciliary contraction, are handicapped and respond to the impulse to convergence only, instead of the impulse to convergence and accommodation. When we consider that vision at the reading distance is demanded by the majority of the human family hundreds and thousands of times daily, the final effect of such an insufficient innervation may be readily appreciated. After months or years a distinct loss of impulse results and exophoria appears. According to this reasoning the degree of exophoria has a distinct ratio to the grade of myopia, and, other things being equal, this is the true state of the case. As we will see in the chapter on exotropia (or manifest external squint), more than fifty per cent. of the cases are found in high myopia. Indeed, it is rare to find equilibrium of the ocular muscles and still rarer to find esophoria among myopes, for the same reason that esophoria is common among hyperopes.

The tests for adduction and abduction show less of the former and more of the latter than in emmetropia or hyperopia, but they do not indicate in which set of muscles the fault lies. Let us assume that in a given case adduction = 10°, abduction = 10° at 20′. It will be seen that adduction is less and abduction greater than normal, and at first thought it would seem as though the power of abduction had gained and was the cause of the tendency outward; but, after considering the lowered adduction, we feel equally justified in ascribing the exophoria to weakened convergence and in stating that the extra abduction represents only lost adduction. If, however, one's figures should read adduction 20°, abduction 10°, we may logically conclude that we are dealing with an excess of divergence, and this conclusion is justified when abduction reaches an abnormally high degree and adduction remains of average strength. Again, we see cases in which convergence power is absolutely *nil*, and which must be regarded rather as a loss of power of coördination from some unknown intracranial microscopic change in the center for ocular coördination, than as a simple loss of convergence. For example, a young woman has recently submitted to two operations for advancement, one on each internus, and although rotary power is complete she cannot overcome a single degree of prism, base out, at any distance from a small point of light. She has never had diplopia, but is a subject of a decidedly diminished capability for fusion when diplopia is artificially produced.

1. Diagnosis.—With a prism of 8° placed base down before the right eye the patient gazes at a candle-flame 20 feet distant. The upper image is the false one and belongs to the right eye. It is not directly above the true, but is above and *on the left*. The prism, base in, that brings the false directly above the true image is the measure of the exophoria.

2. Maddox Rod.—If held horizontally before the right eye, it will produce a long, vertical streak, of the color of the test-flame, to the left of which it passes. The prism, base in, that will carry the streak directly through the flame represents the degree of the defect.

3. The Convex Spherical.—The test-flame, viewed through a strong convex lens, is seen as a large, indistinct, blurred image by the eye before which the lens is placed, and the clear, defined image of the candle will be seen by the other eye in the periphery of the blurred light-area, or entirely separate from it. In exophoria, if the lens is held before the right eye, the clear image will be on the right side of the blurred light, the prism degree of its separation from the center of the blurred image indicating the amount of exophoria.

4. The Parallax Test.—The patient gazes at a light or some small brilliant object twenty or more feet distant, so situated on a large blank surface that it cannot be compared with any other objects in that particular space. A cover is held before one eye, the right for instance, and then suddenly carried over to the left eye, liberating the right, when the image of the light will seem to the patient to shift to the left (*i. e.*, in the direction that the cover is moved), if exophoria be present. This manœuvre must be repeated several times, and the patient's attention perseveringly directed to the way in which the light seems to shift from side to side as the cover is shifted. The prism, base in, that checks all apparent movement of the light noticeable to the patient, will be the measure of the exophoria.

5. Cover Test.—As in the preceding test, the patient gazes at a small distant object. The right eye is covered and, after a few moments, the cover is carried to the left, when the right eye will be seen to move in toward the median line, because, while under the cover, fusion is destroyed and the right eye rolls out into the position most restful for it. If now the cover be carried back to the right eye, the left eye will make a similar excursion toward the median line for the same reason, and when fusion is restored, the return from its divergent position to that of coördination can be readily seen, and measured with prisms, base in. This is simply an objective method of conducting the preceding (parallax) subjective test.

6. Cobalt Glass Test.—In high-grade exophoria, diplopia will be produced by holding a cobalt blue glass in front of one eye, the image seen by which will be blue-red and smaller than that seen by the uncovered eye; the cobalt image will lie on the side opposite to the eye before which the glass is placed, the separation depending on the grade of exophoria, expressed in terms of the prism, base in, necessary to fuse the cobalt and the true image. Defects of less than 3° are, as a rule, not appreciated by this test.

7. A Maddox Double Prism is placed in the trial frame, with the line of the bases horizontal. If held before the right eye, the two images seen by that eye will be on the left of the single image seen by the left eye. That is to say, the images are crossed, and the degree of the defect will be represented by the prism, base in, that brings the three images into the same vertical line.

8. For the Reading Distance, the test by Graefe's dot and line (or, as we prefer, the dot alone) is employed. A prism of 8° is placed, base up, before the right eye, and i the gaze be directed to a large blank card with a 2 mm dot in its center, held 15 to 20 inches from the eyes, tw dots will be seen. The lower dot belongs to the right eye and in exophoria will be found below and to the left o the upper dot, the deviation ranging from 4° to 8° mor than the exophoria found for 20 feet, and measurable b prisms, bases in.

In all cases of exophoria, the various tests should co roborate each other closely both as to kind and degree of defect. Under such circumstances no difficulty whatever should be experienced in arriving at the conclusion that the visual axes have a tendency away from each other. Here again, much as in esophoria, we are dealing with the power of divergence as related to that of convergence, and (excepting a few anatomical cases) not with the externus of either or both eyes, but with the diverging power of the externi, the superior, and possibly the inferior obliques, as opposed to the converging power of the other three muscles of each eye.

Treatment.—After the diagnosis has been firmly established by repeated testing, and due consideration given to general causes underlying the muscular error, in order to determine whether the exophoria is *permanent* (demanding special measures for its relief), or *transitory* (dependent upon loss of nerve-energy from chronic disorder of the nervous system or convalescence from enfeebling affections), one is confronted with the serious question: How shall the tendency to abnormal divergence be overcome? and what are the means best calculated to give speedy and certain relief? The questions have been answered differently by different writers. It shall be our aim to so express the convictions born of considerable experience that the student may be safely guided in his choice. To this end the same general plan will be followed as in the discussion of esophoria, namely: the division of exophorias according to their degree. Parenthetically, it may be stated that many persons who are the subjects of moderately high grades of defect have no symptoms that can be traced to the muscular incoördination, and others again suffer severely from so-called slight defects. In no case is prism or operative treatment to be advocated unless the indications are significant.

1. The Correction of the Error of Refraction.—This reiteration may become wearisome, but the authors concede that the great proportion of muscular insufficiencies are directly or indirectly originated by ametropia, and believe they would be remiss in their duty unless they insisted on every opportune occasion that treatment should be invariably inaugurated by correction of any optical defect that may be present, for the double reason that the optical defect may be the sole source of the muscular symptoms and its correction may prove the cure of the same; further, no restoration to equilibrium can be expected when the cause thereof is acting.

The anomaly under consideration is more frequently found in association with myopia than with hyperopia. The explanation, already given in part, is found in the incomplete or altogether neglected use of the accommodation. The myopic eye has a far point inside of infinity, its distance from the eye depending upon the diopters of myopia. Thus in M. of 3 D. the far point is 13″. This is a convenient distance for reading and no accommodation is required. In M. of 5 D. the far point is 8″, which is too near for the exercise of accommodation. In such cases, therefore, convergence is deprived of the usual added stimulus of the ciliary muscle, and gradually weakens. In the highest grades of myopia, where sustained binocular fixation is impossible, the loss of convergence, as a rule, becomes permanent and exophoria passes into the higher defect, exotropia. It can be readily seen, therefore, that in the presence of myopia the correction of the refraction is a necessary part of the treatment of the muscular anomaly. Indeed, when the myopia is not too high or the patient too old, it is often the only treatment demanded. The correction, in order to be effective, should be worn for near as well as for far. Having thus restored the eye to the condition of emmetropia, and demanded by the use of the glasses the exercise of the normal amount of ciliary contraction in all close use of the eyes, for say two months (in order to test the curative action of the glasses) if it is found that equi- ibrium is not obtained and the symptoms persist—we take the next step in the treatment.

2. **Internal Administration of Nerve Tonics** in large doses combined with prism exercise of convergence. The remedy that has been most warmly endorsed, and that the authors have found extremely serviceable, is the tincture of nux vomica. Strychnin, the main constituent of nux vomica, is for some reason less efficient. The tincture should be administered in increasing doses, commencing with 15 drops thrice daily, increasing every few days by 5 until 45 drops thrice daily are exhibited, or until symptoms of physiologic action of the drug appear. (de Schweinitz.)

3. **Convergence Training.**—The range and power of convergence can be distinctly increased by regular prism exercise. Prisms, bases out, have been used in various ways by different workers for many years for exercise of the adduction: Risley, Michel, Savage, Noyes, Gould and others. (Acknowledgment is gratefully made by the senior author to his friend and preceptor, Dr. Wm. Thomson, Emeritus Professor of Ophthalmology in the efferson Medical College, for instruction nearly twenty ears ago in his office, in the use of prisms both as method of stimulating the innervational force of the xternal ocular muscles and as a means of relief in mus- ular asthenopia.) The battery of Noyes or Gould is a convenient arrangement of prisms and can be em- loyed with benefit, although the costliness of such pparatus limits its use to the consulting room. For ome use the prisms are to be mounted either in an ordi- ary spectacle frame or in one of the numerous frames

specially devised for carrying square prisms that may be increased in strength *ad libitum* (see Fig. 19). These have come into general use largely through the persistent advocacy of Gould, who bases the following method on the idea of innervational abnormalism as the cause of much heterophoria: The strongest prisms, bases out, with which the patient can fuse a small object or point of light at 20 inches, are placed in a spectacle frame. While the patient fixes on the object, it is carried rapidly across the room (or the patient backs away from the light across the room), which ought to be at least 20 feet long. The patient then closes the eyes or lifts the prism-glasses while the fixation-object is brought back to a point 15 to 20 inches from the eyes, when the manœuvre is repeated. This exercise is to be performed several times daily for from three to five minutes each time. It will often require the strictest injunctions on the part of the surgeon to impress upon the patient the necessity for, and the importance of, these calisthenics, and to bring him to realize that the partial or complete relief of his asthenopia is largely in his own hands. The coöperation of the patient once secured, the prisms can be rapidly increased in strength and the fusion force often carried to 100° inside of eight or twelve weeks, in which event the patient is pretty sure to have experienced marked relief from the asthenopia. The main difficulty in this method lies in the persistence necessary to convince the patient that little is to be hoped for without the heartiest coöperation. Many cases of severe muscular

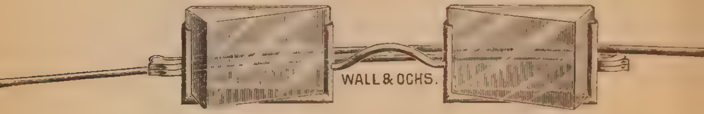

FIG. 19.—Trial frames for prismatic lenses.

asthenopia can be made quite comfortable by the combined action of increasing doses of nux vomica and prism exercise; indeed their value has been universally acknowledged. An interesting clinical fact in connection with this method of treatment is that if a patient start with an exophoria of 10° for distance and 18° for 20 inches, the asthenopic symptoms may be entirely dissipated by nux vomica and prism exercise for two or three months, notwithstanding which the exophoria for distance will remain at 10°, while that for near may have fallen to 3° or 5°, or have entirely disappeared; all of which goes to show that exercise of the adduction may increase the range of convergence (as can be found by prism tests of the adduction and abduction), but will seldom influence to any extent the muscle-balance for 20 feet. In other words, we have simply made easy to the patient a much-needed coördination, and have perhaps at the same time trained the cortical fusion centers to a higher degree of efficiency.

No case should be permitted to go to operation until these measures have been faithfully tried. They are peculiarly useful in individuals who have passed through a debilitating illness, such as typhoid fever, la grippe, measles or diphtheria; also in young and growing subjects; and their action is always enhanced by life in the open air, general muscular exercise, and nutritious diet.

4. **Prism Correction.**—When the exophoria is of such grade,—differing in different individuals,—and the symptoms of such severity that relief can be obtained only by relaxation of the effort of convergence in maintaining equilibrium, the wearing of prisms in the position of rest is recommended. The objections to this method of treatment, alluded to under the use of prisms in esophoria, obtain in the correction of exophoria. Hope or prospect of cure is practically abandoned. In a few exceptional

cases only can the glasses be subsequently laid aside without a return of the discomfort relieved by their use. While the prisms are worn the visual axes diverge from each other and the globes assume that position in the orbits most restful for them. The effort of convergence is in abeyance and the strain of opposing the stronger power of divergence is relaxed. The eyeballs are diverged and single vision is maintained by the prisms. For these reasons it is good practice to allow a small proportion of exophoria to remain uncorrected by prescribing prisms that permit constant but limited exercise of convergence. Thus in exophoria of 8° for near, prisms of 3°, base in, before each eye may be ordered. Whether or not the prisms should be worn constantly depends upon the degree of exophoria and the symptoms arising from it when the eyes are used for distance. Almost without exception there is a greater degree of exophoria for the reading distance than for 20 feet, although the ratio of increase has not been stated and must depend in large measure upon the peculiarities of each individual. Defects of less than 4° at 20 feet rarely require the use of prisms for that distance. When higher than this, the symptoms will usually demand their constant use. Also, it may be necessary to provide stronger prisms for reading than for distance. There is a limit, however, beyond which no practical benefit arises from prismatic correction. The limit varies according to circumstances of sex, age, occupation, etc., of the patient, but it may in general terms be placed at 5° before each eye. When prisms fail to afford relief, we advance to the next step:

5. Operation.—The surgeon has the choice of tenotomy of the externi, or advancement or re-section of the interni. If exophoria is that condition of outward tendency of the visual axes arising from and dependent upon a loss of convergence, there would seem to be no alternative between these two radical procedures; yet the choice must invariably fall upon measures which will strengthen convergence rather than weaken divergence. Thanks to Landolt's persistent labors, the advocates of tenotomy in exophoria are becoming constantly fewer and the followers of advancement or shortening more numerous. In our own practice, tenotomy of the externi for exophoria and functional exotropia has fallen almost into disuse. We believe this is correct practice founded upon sound physiology, yet we are obliged to admit, in all fairness, that our results have not been always as satisfactory as we and our patients could wish. The operation is far more difficult and tedious to perform than tenotomy, and more painful for the patient, and, moreover, it may not increase the power of convergence. When accurately done we believe it is superior to and gives more lasting results than tenotomy. In considering the treatment, the etiologic relation between hyperphoria and exophoria must be constantly borne in mind. It is our conviction that not infrequently correction, by tenotomy or otherwise, of the former, will cure the latter, and that Stevens is correct in referring inconstant and varying degrees of want of lateral equilibrium to abnormal vertical tensions. It is in such complicated cases that the test by the cobalt glass possesses advantages superior to most of the others, for in the presence of diplopia the false (cobalt) image will designate in every instance the new direction taken by the now deviating eye. When either defect (or both) requires direct treatment, attention should first be directed to the restoration of vertical equilibrium, and only after the lapse of some weeks to the lateral tendencies, should they continue abnormal. Again, the tensions may be so modified by paralysis of accommodation, (induced to determine the refractive error), that the diagnosis that seemed justifiable upon examination without mydriasis can not be sustained. It is not uncommonly found that both esophoria and exophoria will increase under mydriasis. The former, because the convergence stimulus is no longer regulated by accommodation stimulus, and in the effort for clear vision the tendency inward may become a real turning. The latter, because convergence, already weak, becomes still more so upon withdrawal of the stimulus of accommodation. These statements may seem contradictory since the one explanation is offered for two opposite conditions, but they are the result of many observations and are physiologically plausible.

The limitation of the operation of advancement to one internus, or the inclusion of both, is to be decided on the merits of each case. The number of prism degrees of convergence gained by a single operation depends largely on the preserved power of adduction. As a rule, it does not exceed 5° or 6°, and when more than this amount of correction is demanded, as is the case in most subjects for operation, each internus should be advanced. It is admitted that higher degrees of relaxation of divergence can be obtained by full tenotomy of the externi, and at first sight this operation would appear to be indicated, but its apparent advantages are outweighed by the serious objections of diminished external rotation, want of permanency in the result, and only relative and inconstant increase in adduction. It is our opinion that the best results are to be obtained by advancement of each internus, dividing the amount of correction between the two eyes, as nearly as may be, at the same sitting. Postponement of the second operation is not contraindicated, yet the preference should be given to the double operation since, by this plan, the dread and anxiety of the patient are reduced more than one-half, and the eyes are in a con dition to respond equally to the innervational response. If these facts are explained to the patient it is improbable that he will raise objections to the plan proposed, but will submit philosophically to the reasoning of the surgeon.

Section V.—HYPERPHORIA.

Hyperphoria is that condition in which there is a tendency of one visual line in a direction above that of the other, *notwithstanding the presence of perfect binocular vision.* Hyperphoria (latent vertical squint) differs from hypertropia (manifest vertical squint) only in that single vision is not possible in the latter condition, and the eye that is more ametropic, or is at the greater muscular disadvantage, gives up the struggle and actually deviates upward or downward.

If we accept the above definition, it will be found that hyperphoria is by no means rare, nor is its presence inconsistent, in some cases, with perfect health and comfort in using the eyes, in which event there can be no excuse for treatment of the tendency to deviation. An analysis of the findings at the first visit in 100 consecutive refraction cases occurring in our practice, shows the frequency of hyperphoria under such conditious to be 30 per cent. Carpenter found it in 35 per cent. of his private cases at the first consultation; so it seems safe to assume that fully one in three of the patients who are subjects of muscular or accommodative asthenopia will, during the preliminary examination, reveal some degree of vertical imbalance, either alone or in association with an esophoria or exophoria; yet many of these will demand no other treatment than painstaking correction of the ametropia under complete paralysis of the accommodation. This class of cases (ametropic), along with those secondary to gout, rheumatism, central nervous lesions and other general diseases, comprise what are known as *temporary* hyperphorias in contradistinction to true or *permanent* hyperphorias which occur independently of constitutional conditions, and persist after long use of the closest correcting lenses. Such hyperphorias are commonly due to overaction on the part of one muscle (hyperkinesis) or underaction on the part of another (hypokinesis). They may also result from anomalies in the formation and relative position of the orbits, or from peculiarities in attachment, development, insertion, or action of one or more muscles.

It is, therefore, obvious that in tracing a given hyperphoria to its origin there is a large array of factors that must be individually weighed before rational treatment can be pursued.

The symptoms of hyperphoria are ocular and reflex. The ocular symptoms are:

1. Chronic hyperemia of the lids, often giving rise to a condition aptly likened by one writer to the "hot eye" of gout.

2. Epiphora, generally unilateral and seemingly without nasal or ocular cause.

3. It is not unusual to find vision of but $2/3$ to $1/2$ in an hyperphoric eye even after accurate correction of a low-grade ametropia; indeed, it may be impossible to give the patient in each eye better vision than $\frac{5}{7\frac{1}{2}}$, a fact easy of explanation, however, if it is borne in mind that an actual vertical imbalance of 1° will result in a separation of images at 20 inches of 6½ mm. Such a patient, if a worker at the occupation distance, will most surely be troubled from time to time with

4. Confusion of images, which is the result of the transient diplopia. It may be urged that many students carry a hyperphoria of 1° or more through a busy career extending over many years without experiencing any annoying

symptoms whatever. Such persons either possess a physical and nervous system that would weather any storm, or they have, consciously or unconsciously, learned, by carrying the head toward one or the other shoulder, to neutralize their hyperphoria wholly or in part. This habit constitutes the fifth important symptom of true hyperphoria, namely:

5. *Carriage of the Head.*—The head is usually tilted toward the shoulder opposite to the hyperphoric eye, a statement that may sound strange, but when it is remembered that in right hyperphoria the image is really seen lower by the right eye, it naturally follows that the head must be tilted toward the left shoulder if the images are to be brought to a level and binocular vision thus rendered an unconscious act. Graefe referred to the turning of the face to one side in "insufficiency of the interni" to aid the weak adductors, but he said nothing of the peculiar carriage of the head in hyperphoria, which is equally common.

A peculiar facial expression will often be noticed, especially when an hyperphoric is in animated conversation; several furrows will ridge themselves above one eyebrow, and even the eyebrow itself may be raised above the level of its fellow from 4 to 10 mm., giving a quizzical expression to the face. There should also be mentioned, in this connection, the wide open eye, or stare, seen in many hyperphorics. Occasionally an apparent ptosis of many years' duration is dispelled by prismatic or operative correction of hyperphoria.

Not less important, as immediate symptoms of vertical deviation tendencies, are the painful eyes, photophobia and drowsiness induced by any long-continued near work, notwithstanding that the patient is wearing proper lenses.

The reflex symptoms of hyperphoria include more or less constant headaches, amounting sometimes to a migraine, nausea, vomiting, dizziness, and vertigo. In some instances the latter is so marked as to cause momentary unconsciousness and a symptom-complex that has more than once led to a diagnosis of epilepsy or even cerebral tumor. We believe it is the latter class of cases that are vaunted by many as instances of essential epilepsy partially or even entirely curable by eye-treatment alone.

The headaches of hyperphorics are almost invariably aggravated by near work; also by moving in a crowd or watching rapidly-moving objects (panorama headache of Bennett). Other hyperphorics will escape the headaches, but present, instead, frequently recurring nausea, vomiting and vertigo, the latter especially brought on by any continued looking up or down.

Diagnosis.—The assertion that hyperphoria exists in a given case ought not to be based on any one test, but demands corroboration by every test at our command. Moreover, no problem in ophthalmology calls for greater accuracy and adjustment of instruments, and closer observation. For conducting tests, Risley's or some similar optometer (Fig. 20) armed with two rotary prisms, gives the most satisfactory results. The 30° rotary prism should be fixed with base apex line vertical for testing the lateral muscles, and the 10° prism with its base apex line horizontal for testing the vertical ones.[8]

When the patient's face has been brought close up against the back surface of the optometer, and the latter been found to be absolutely level by means of the plummet or spirit level attached for the purpose, the compound

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[8] Jackson's 10° rotary prism, composed of three different prisms, is preferred because the markings in its lower powers are widely separated, and great accuracy is thus secured.

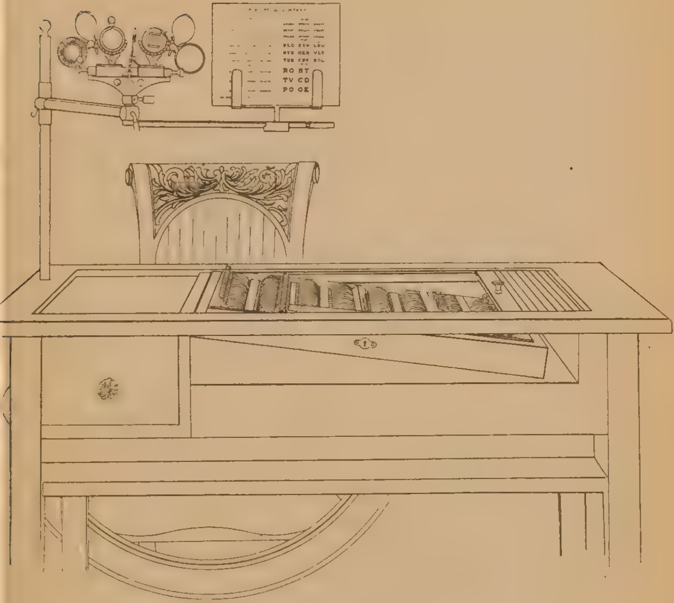
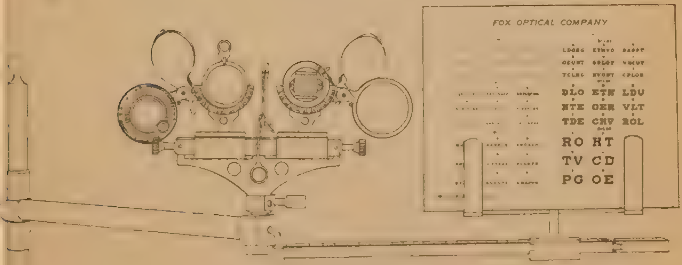


FIG. 20.—Optometer table.



Enlargement of optometer.

Maddox rod is slipped into one of the cells, say the right one, and so turned that the streak of light is exactly horizontal. If the streak passes above the flame, there is shown either right *hypo*phoria (downward tendency of right eye), or left *hyper*phoria (upward tendency of left eye). If the streak be found below the flame there is right *hyper*phoria (upward tendency of right eye) or left *hypo*phoria (downward tendency of left eye). The same facts hold good when the Maddox rod is placed before the left eye, so that the test can be applied by placing the rod before either eye. The prism that carries the streak into the flame is the measure in degrees of the hyperphoria.

Diplopia or Phorometer Test.—Place a 12° prism, base in, before either eye, and before the other a colored (red) glass to assist the patient in differentiating the two images. Be sure that the prism has its base-apex line absolutely horizontal, (for if a 12° prism be rotated but 5° from the horizontal, there results 1° of vertical displacement, and the 1° of hyperphoria thus produced by bad adjustment may lead the observer into serious error). With the prism properly placed, if one image is lower than the other, there is upward deviation (hyperphoria) of the eye to which that image belongs, which, as in the findings with the Maddox rod, is to be measured in the terms of the prism that brings the two images to the same level. If the phorometer be used, the prism or prisms with which diplopia is affected are already in position, and if one image be higher than the other, all that remains to be done is to rotate the prisms by means of the small lever until the images are level, when the resultant right or left hyperphoria will be indicated by the pointer on the arc graduated in degrees on the front of the phorometer.

Parallax Test.—Seat the patient 20 feet or farther from some small object that is situated directly in front of and on a level with the eyes. The object (a small luminous point of light in the middle of a large black area, for instance) must be so placed that it will not be projected upon any surface back of it. Note carefully that the patient's head is not tilted to one side, then carrying some sort of cover to and fro several times from one eye to the other, the patient is requested to watch closely whether the object as seen by one eye appears any higher than the other. If so, vertical imbalance is present and its degree is to be determined by the prism that will make the object appear on the same level with each eye. It will sometimes be necessary to carry the cover from one eye to the other 15 to 20 times before any difference in level in the apparent position of the object becomes noticeable, but by this means the lower degrees of hyperphoria are frequently unmasked. The parallax test, when thoroughly understood, is susceptible of great delicacy of application and by it ½° of hyperphoria is easily detected. Duane, whom we must thank for the test, claims that he thus estimates **even ¼° of hyperphoria.**

If the patient be ametropic, he must wear his correction during all the tests above mentioned. Thus far we have determined only that one ocular axis has a tendency to deviate above, or the other below the axis of the fellow eye. We have not located the defect nor decided whether it be due to overaction of the muscles of one eye, or underaction of the corresponding muscles of the other eye. Resort must now be had to prisms, to learn the prism-power of upward and downward rotation. This will be found to fluctuate from 2° to 3°, but the fact of prime importance is, that whatever the power of prism-rotation, supraduction and infraduction should be equal. If the eyes can overcome a 3° prism, base down, they should likewise overcome a 3° prism, base up. A difference of 1° or more between the supraduction and infraduction of the same side indicates the probable existence of hyperphoria; moreover, a supraduction exceeding 3° (except in moderate and high myopia) is always suspicious and presumptive of hyperphoria.

If there be suspicion of hyperphoria and the supraduction and infraduction as found by prisms prove equal, it becomes necessary to map out the field of monocular fixation, especially the upward and downward limits. This is done by having the patient follow with the eye (but not with the head) a very small test-object, such as a black dot on a white card, which is carried as far up and as far down as the eye will follow it without wavering or receiving a blurred image of the object. The arc through which the eye has rotated may be measured either roughly by the eye of the observer, or approximately by some form of perimeter, the head resting on the chin-support and the fellow eye excluded as in ordinary perimetry. This examination should show at least 35° to 38° of upward, and from 50° to 55° of downward rotation. For the precise measurement of these rotations, Stevens employs his tropometer (described above), and if upward rotation falls short of, or exceeds, 35° to 38°, or if downward rotation exceeds or falls short of 50° to 55°, the corresponding elevators or depressors are to be viewed as causative in the hyperphoria. If upward rotation is excessive, the hyperphoria is likely due to overaction on the part of the elevators of the hyperphoric eye. If downward rotation be excessive, the hypophoria is likewise due to excessive action (hyperkinesis) of the depressors of the hypophoric eye. If upward or downward rotation be insufficient (hypokinesis), the deviation is probably due to weakness, or even paresis, of one or more of the vertical muscles, in which case diplopia can be readily elicited when the eyes (but not the head) are turned 20° or more from the primary position, the diplopia increasing as the candle is carried in the direction of action of the weak muscles.

Finally, when the tests for hyperphoria at 20 or more feet are concluded, search should be made for vertical deviation tendencies at 15 to 20 inches with Graefe's dot-and-line test. It will be found that hyperphoria for near is always a trifle more than hyperphoria for distance, a point of no small significance in applying treatment to these cases.

We would suggest, in this test, the use of the dot only, or, better still, a 1 to 2 mm. perforation in the center of a large black card illuminated from behind with a small incandescent electric light. This will also permit coloring of the double images with the cobalt and red glass, and the ease with which the patient can then differentiate the two objects will lend much to the accuracy of the test. True, the conditions are not those under which the eyes work ordinarily, and this fact must be given due allowance; nevertheless many valuable data are thus brought to light.

The Relation between Hyperphoria and Lateral Deviation Tendencies.—It has been claimed that many cases of esophoria and exophoria are directly dependent upon and caused by a tendency to upward deviation of one of the visual axes, and no doubt there is a large grain of justice in this claim. It is easy to conceive that when vertical tension is out of equilibrium the lateral muscles in their efforts to fuse images will tend to turn the eyes now in and now out, or will acquire a habit of forcing the lateral tendency in one or the other direction. This dependence has been illustrated by comparing the lateral muscles, in their efforts to fix, with the lifting of a log with a pair of tongs, the clamps of which are not in a horizontal line. Many cases have been reported where not only [illegible] headaches, but general [illegible] maladies have been cured by correction of vertical anomalies, and in the consideration of causes which lead to esophoria or exophoria, vertical troubles should be given a prominent place. Theoretically, all cases of uncorrected hyperopia ought to have upward and inward tendencies on account of the resultant action of all the external muscles supplied by the 3d nerve, and the reverse will hold in myopia, namely, a tendency downward and out. This inter-dependence can be well shown where the prism of the [illegible] is kept [illegible] in place of [illegible] with the cobalt glass. It will be found by this test that there are but very few lateral turnings that are not complicated with vertical turnings, and it will also be found that hyperphoria frequently changes from one eye to the other, according to the eye used in fixation. If the right eye should fix and the left be covered with the cobalt glass, then the blue image to the left and below, we have left hyperphoria and exophoria while the cobalt glass is before the left, and if the glass be transferred to the other eye, that eye may pick up the blue image, and we have R. hyperesophoria. In other words, the cobalt glass determines the fixing, and hence, also, the deviating eye. The reason for the association of hyperphoria with esophoria in hyperopia has already been explained, and by analogous explanation the the association of hyperphoria with exophoria. If we admit that the tendency outward is a passive anomaly and due to a loss of convergence, the deficiency of innervation affects not only internal but also vertical turnings, and where we would have an exophoria hyperphoria, we should expect to find it complicating exophoria.

Therefore, in considering the treatment of esophoria or exophoria, the condition of the vertical muscles must

must be employed with great caution. The persistent use of such corrections will suffice in most cases, if not to entirely dispel the hyperphoria, at least to render the patient so comfortable that treatment of the deviation-tendency, as such, need not be considered. If, after faithful use of the correcting lenses and careful attention to dietetic and other conditions, the hyperphoria persists and is annoying, it will be admissible to try the effect of a prism or prisms that correct about two-thirds of the hyperphoria. For convenience, these prisms may be slipped into what is known as an extra front to be hooked over the regular glasses (Fig. 21). The prism effect may be divided between the two eyes (base down before one, base up before the other), or the whole prism desired may be used, base down, before the hyperphoric eye. By this means, prompt and most astonishing relief is sometimes obtained, as, for instance, in the following case:

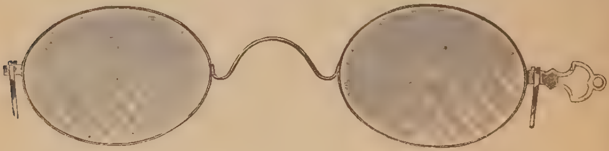


FIG. 21.—Extra front.

F. E., aged 20, a goldsmith, works all day at a distance of from 8 to 10 inches soldering small gold trinkets. Given 18 months ago by a colleague (who used a cycloplegic) R.E.+.50 D+.50 Cy 90°. L.E.+0. 75 D.S. With this correction he was much more comfortable at his work for two or three months, but eyes and head began to trouble again, when his oculist, after much pains, ordered a ½° prism, base down, to be incorporated in the left glass, indicating that he found *left hyperphoria.* (This point is emphasized because of the subsequent developments in the case.) Again he pursued his work with renewed comfort for about three months, when the old train of eye and head symptoms once more took up its march. At this time he came under our observation, and on September 14, 1897, he was carefully refracted under thorough cycloplegia and the findings of the previous worker were confirmed in all but the muscular details.

September 16, 1897. Maddox rod shows esophoria ½°. No hyperphoria. Abduction = 7°. Adduction = 15°. Left supra- and infraduction = 3°.

September 30, 1897. Maddox rod used over patient's correction shows exophoria 1°. *Right* hyperphoria ½°. (*Left* hyperphoria had been found by his former adviser). With the parallax test R. hyperphoria 1½°. Exophoria 2°.

October 14, 1897. Left hyperphoria 1° full, with parallax test! Maddox rod shows vertical balance. Control tests made four times during the succeeding month showed R. hyperphoria 1° full with the parallax test, and each time R. supraduction equalled 4½°, while R. infraduction equalled 2½°.

This accord in results in four consecutive examinations justified having the ½° prism, base down, taken out of the correction of his left eye, and having him try ¾° prism, base down, before the right eye in a slip front to be hooked on over his glasses while at work. One month later he stated that he had never been so comfortable. He was then directed to have the ¾° prism, base down, incorporated into his right glass, because he was so much annoyed by the reflections from the prism in the extra front. Four months later the patient stated that he was working hard at his soldering bench (at a distance of 8 inches) for 10 hours a day, without discomfort of either eyes or head.

This case illustrates how the most careful worker may be led into error by a spastic hyperphoria which we believe this young man to have shown in the earlier part of his trouble. Later, long-continued observation of his case and prism estimation of the upward and downward rotation showed not only right hyperphoria, but the difference between the right supraduction (4½°) and infraduction (2½°) pointed to the overacting elevators of the right eye as the cause of the deviation. Theoretically, tenotomy of the right superior rectus or of the corresponding inferior oblique was indicated, but prisms in the position of rest (favoring the underacting or weaker depressors of the right eye) gave the patient such prompt and lasting relief that operative measures were not to be thought of. It is to be remembered that hyperphoria for 15 to 20 inches is always greater than for distance. It is, therefore, good practice when ordering reading corrections for hyperphorics to have incorporated into them the prism that represents the full hyperphoria for 20 feet, while no more than two-thirds of the prism correction for distant hyperphoria is usually ordered, especially when prisms are being prescribed for the first time. Yet warmly as we wish to endorse the practice of temporary trial of prisms in the hook front, there are many individuals whose symptoms are only aggravated by this measure, in which case exercise of the vertical muscles may be tried by means of prisms placed base up or down, as the case requires. Savage believes this method to be of value in deviation tendencies under 1½°, and says: "The prisms used should range from ¼° to 2°; most cases will not require stronger than a 1° prism. The apex of the prism should be placed in the direction of action of the muscles to be developed, the patient exercising from 2 to 10 minutes, two to five times daily. The object looked at must be 20 feet distant, and it should be seen *through* the prism

5 seconds, and then *without* the prism 5 seconds, and so on throughout the sitting." While our own experience with this method in hyperphoria has been limited, our results have not been such as to lead us to expect much relief from this kind of training.

If then, a patient presents persistent hyperphoria and will tolerate neither prisms in position of rest nor exercise of the weaker muscles, are we to turn immediately to operation as a last resort? Not so. Many factors must be determined before operative treatment is applicable. For instance, it is not to be thought of when the hyperphoria is recent, progressive, or variable; in case of central nervous disease; in rheumatic, gouty or diabetic subjects; or in those who have not enjoyed binocular vision for years (strictly speaking, these are instances of hyper*tropia*). Moreover, when hyperphoria complicates esophoria or exophoria, it is not infrequent for the vertical anomalies to right themselves, or at any rate for the symptoms attributed to them to disappear when the lateral muscles have been well trained with prism gymnastics. Hence we find operative treatment of the hyperphorias narrowed down to the permanent, constant or static variety, where the deviation tendency is constant in amount and character, and where all tests agree in showing marked over- or under-action of one set of elevators or depressors.

In the former class of cases (overaction) tenotomy is highly satisfactory and often brilliant in results, if the case has been selected in accordance with the above suggestions. The dissipation of reflex symptoms following upon tenotomy for hyperphoria is sometimes little short of wonderful, and has doubtless led many witnesses of such cures into operative treatment of hyperphorias that are distinctly outside of the operative class, and given the extreme conservatives in ophthalmology occasion to deride the judicial and the unjudicial alike as "muscle-snippers."

Hyperphoria, constant in degree and kind, due to underaction on the part of the vertical muscles, is best met by advancement or muscle-shortening of the underacting muscle or muscles, and in cases properly selected, the same gratifying results may, as a rule, be expected.

Operation, whether tenotomy or advancement, should always aim to slightly under-correct the deviation, the remainder being often amenable to prism exercise of the weak muscle immediately and from 24 to 48 hours after the operation; in this way the effect of an operation that is insufficient may be considerably increased; any slight remaining deviation can be readily met by the necessary prism in the position of rest.

Surgeons of large operative experience with muscular anomalies prefer to aim at a slight over-correction of the imbalance, because, as they assert, reattachment of the operated muscle is almost sure to be attended with some loss of the original effect. The practice is a safe one, however, only in the hands of those who have perfected themselves in the technique of operations on the ocular muscles.

Section VI.—CYCLOPHORIA.

Some surgeons believe that the real nature of certain rebellious cases of eye-strain lies in a want of equilibrium on the part of oblique muscles. As yet we are not in possession of sufficient figures to indicate how frequent the condition and what its relation to the various anomalies of refraction may be. Attention was first directed to this phase of muscular imbalance by Savage, in 1892, and termed by him "insufficiencies of the oblique muscles." The condition is easily detected by covering one eye and placing before the other a Maddox double prism with its base line horizontal, and having the patient look at a straight horizonta line drawn on a large white card held 18 inches away

The line appears double. The covered eye is now freed, when a third line will come into view midway between and parallel with the other two if all the muscles are in equilibrium. If there is no vertical or lateral imbalance, and the oblique muscles are abnormal in their tensions, the middle line will be tilted up or down at one or the other end, according as the superior or inferior oblique is at fault, and further according as the right or left eye be tested. The eye that has no prism before it is the one which is being tested. The position of the line when the different oblique muscles are faulty in their action is shown in Fig. 22. This test may be modified by using a thin horizontal slit two inches long in a large black card behind which is held a small electric light. The streak in the card thus becomes luminous and may be colored differently for the two eyes by slipping cobalt or red glasses into the trial frame, enabling the patient to distinguish the single from the double images much better. The middle line may be nearer the bottom than the top line, or vice versa, indicating coincident hyperphoria, or the middle line may extend farther to the right or to the

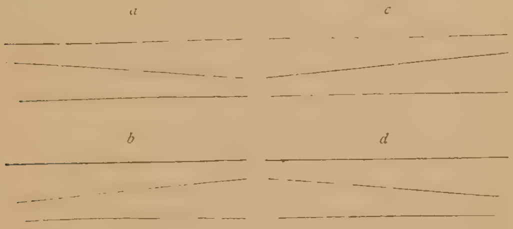


FIG. 22.—Position of the lines in
a. underaction of the left superior oblique muscles.
b. " " " inferior " "
c. " " right superior oblique muscles.
d. " " left inferior " "

left than those above and below, in which case lateral imbalance coexists.

The treatment of cyclophoria consists in what is termed "rhythmic exercise" of the weak oblique muscle or muscles accomplished by the rotation of concave or convex cylinders, preferably the latter. For convenience during this manœuvre, Savage suggests frames made of German silver with rims deeply grooved to allow free rotation of the lenses. "The rims are notched at points 15° apart from 90° to 45° in either the lower nasal or temporal quadrant, depending on the muscles affected. Convex cylinders of about + 1.50 D. are usually employed, moving the axis from 90° out into the lower temporal quadrant for insufficiency of the superior obliques, and from the vertical into the lower nasal quadrant for insufficiency of the inferior obliques, the effect in either case reaching its maximum at 45° from the vertical. The exercise may be begun with a 0.50 D. to a 1.00 D. cylinder and increased each day by a 0.50 D. up to 3.00 D., beyond which it will seldom be necessary to go. The cylinder strength should be divided between the two eyes. Placing the cylinder in the above-mentioned frame with the axis at the notch marked 1, the patient is instructed to look at a candle or gas flame 20 feet distant for 5 seconds, then without the cylinders for 5 seconds and so on, for 5 minutes. The two cylinders are now revolved so that their axes are at notch 2, repeating the alternating periods for 3 minutes, when the cylinders are placed with their axes at notch 3 and the 5 seconds alternating exercise continued for 2 minutes longer, making 10 minutes in all." This constitutes a daily sitting. Experience thus far gained indicates that two to four weeks of such exercise will be required in most cases.

The objection may be with fairness urged against this method of diagnosis that it affords no means of discrimination between an affection of one of the oblique muscles, and an affection of two or more of the recti muscles. In the latter the torsion, which is a resultant of their contraction, would be diagnosed by the double prism in exactly the same manner as would be the torsion induced by the contraction or relaxation of one of the obliques.

Section VII.—PROGRESSIVE HETEROPHORIA.

Little attention has been paid by writers on muscular anomalies (with the exception of Stevens, who has discussed the subject in all its ramifications) to those cases that properly cannot be classified either in the category of 'phorias or 'tropias, but occupy a middle ground between them. Practically, such a distinction is not of the first importance to successful treatment. We recognize the two conditions as successive stages of the same affection dependent upon similar causes and treated upon identical principles. No sharp line of separation may be drawn between the two clinical varieties in all cases. While many tendencies to deviation from parallelism always remain latent, others pass at a later stage of their existence into actual turnings or manifest squints. They are characterized by occasional and momentary diplopia, dependent upon a sudden lessening or withdrawal of the nerve-impulse necessary to maintain single vision, or a transient loss of fusion-force. The degree of the defect is so high that the slightest loss of muscular tone, or decrease of nerve-energy, or excessive work, or any condition of general exhaustion, will be reflected upon the muscles that are all so strained that it is difficult, even under the best of conditions, to maintain binocular single vision. As a result of one or all of these predisposing influences, the weaker set of muscles yields to the relatively increased tension of the stronger set and the visual axes are turned from parallelism. This may obtain either for distance or for near, according to the kind of defect. Instantly that the brain realizes that the visual axes have deviated (the consciousness of which is induced by double images), a renewed impulse of greater force is sent down to the muscles that have yielded, and they are stimulated to increased effort. The diplopia immediately disappears in consequence of this extra stimulus.

To the symptomatology of heterophoria of high degree is added, by this complication, the symptom of momentary double vision or blurring without actual diplopia. The treatment is the same as that for heterophoria, but naturally, in consequence of the high grade of the defect, resort must be had more frequently to operation.

CHAPTER II.

HETEROTROPIA.

Section I.—ESOTROPIA.

Synonyms.—Internal or convergent strabismus; internal squint.

Esotropia is that condition in which the visual axes are directed toward each other, crossing at some point inside of infinity; or, more accurately, the visual axis of one eye is directed toward that of the other. The image of the object engaging the attention of the patient falls upon the fovea of the fixing eye, and upon some portion of the retina to the nasal side of the fovea of the squinting or deviating eye. Hence, the visual axes are not, strictly speaking, inclined toward each other; but the axis of the fixing eye is directed straight at the object, while that of the deviating intersects that of the fixing eye at a point nearer the face than the object; in other words, both eyes are not abnormally converged at the same moment (see Fig. 23). The burden of the convergence is borne by one eye only, transferable

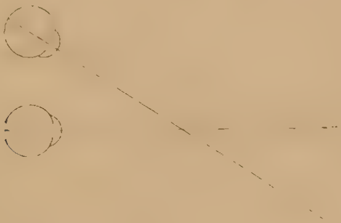


FIG. 23.—Crossing of visual axes.

under some conditions to the other, but never manifested simultaneously in both. This statement is true only of the properly so-called functional esotropias, and excludes paralysis of the external muscles and association paralysis.

Esotropia is monocular (or constant), and binocular (or alternating). In the former, one and always the same eye, under all circumstances, is used for fixation, unless artificially and purposely excluded from the act of vision, when the second eye deviates. In the latter, either eye is used indifferently for fixation, and the squint is instantaneously transferred to the eye not so used.

MONOCULAR OR CONSTANT ESOTROPIA.

Monocular esotropia is characterized by (1) amblyopia of the squinting eye, and (2) invariable fixation of the sound eye.

The origin of amblyopia, or indifferent vision, has been for a long time a subject of contention among writers on ophthalmology. The disputed point is whether its origin is congenital or acquired from disuse. The arguments to prove that it is congenital seem to us to prevail, and we therefore discuss amblyopia among the causes of squint rather than among its consequences. The ophthalmoscope shows no defect in the media, and the details of the eyeground in every particular conform to the normal standard, although the field of vision is not infrequently defective (de Schweinitz). That no minute anatomic changes of the optic nerve occur in the fovea or in the foveal fibers seems clear from the report of several cases in which an amblyopic squinting eye was restored to useful vision in the course of a few weeks by its enforced use. Again, the error of refraction of the amblyopic eye not infrequently differs very little from that of the seeing eye, certainly not enough to account for that grade of defect which will exclude it from all participation in vision. The well-known argument that an eye blind for many years from cataract may reacquire useful vision after extraction of the crystalline lens also seems to disprove the claim that amblyopia can arise from disuse. It is probable that in consequence of a functional, (but not anatomic) defect in the basal ganglia or the cortical centers for vision, acting in conjunction with a degree of hypermetropia high enough to disrupt the association of convergence and accommodation, one eye has been excluded from vision and its function held in abeyance. This statement is not offered as an explanation, but simply as a reason or as a theory which helps to account for defective vision of amblyopics. Whatever the explanation may be, it is true that amblyopia is invariably associated with monocular squint; that it limits the squint to one eye, and stands in the way of a scientific correction of the muscular error, since binocular vision is practically unattainable.

In some cases restoration to parallelism of the visual axes can, in fact, be effected; only for a brief period, however, since the blurred image seen by the amblyopic eye interferes with the clear one seen by the good eye, and in the interest of good vision the indistinct image comes to be disregarded and an internal or external deviation is developed. Amblyopia is incurable, excepting under the conditions above referred to, viz, the destruction of the eye that had hitherto been used for vision. Improvement has been reported as the result of exclusion of the good eye, enforcing education and training by constant use of the other; it has also been claimed as a result of operation; but in our experience the means often successfully employed to correct squint in childhood without operation, such as atropin, bandaging of the good eye, etc., have proved ineffectual in curing amblyopia, and the hope that vision may be restored to a practical and useful extent by these means has not been fulfilled. "Amblyopia" is used by writers and teachers without a true conception of its meaning and of its limitations. It signifies simply "indifferent or doubtful vision," not blindness (for which the obsolete word is amaurosis), and when not qualified by an adjective, such as toxic, renal, etc., indicates simply that the patient's vision is defective from no known causes. The use of the word should be limited to eyes whose vision is lower than $\frac{20}{50}$. An eye is sometimes wrongly called amblyopic. Perseverance scientifically applied will often be rewarded by a decided improvement of vision, by correcting lenses.[1]

Diagnosis.—The methods that take into consideration the *apparent* deviations of the visual axes are unreliable. For example: the angle alpha, made by the intersection of the optic with the visual axis, may be abnormally small, giving rise to an apparent internal squint, while the muscles may be in equilibrium. Again, the interpupillary distance, the shape of the orbits, the size of the commissure and other anatomic peculiarities should always be taken into consideration.

Inspection will show a want of coördination of the visual axes by the fact that the sclera of the temporal side of the squinting eye is exposed in greater extent than that on the nasal side; further, that the cornea is deflected toward the nose.

Cover Test.—In permanent internal squint, one eye, as has been said above, fixes and the other deviates. Now, if the fixing eye is excluded from participation in the act of vision by a screen placed before it, the cornea of the deviating eye will be seen to promptly make an excursion outwards, the degree of movement varying according to the degree of the squint. In other words, the hitherto squinting eye now attempts to fix by so rotating the cornea that the rays of light passing through it will fall upon the foveal region and the squint is transferred from one to the other eye. The degree of squint can be estimated with a fair degree of accuracy by interposing prisms with their bases out, increasing in strength until all outward movement of either eye is abolished. In the absence of a high grade of amblyopia, fixation is sufficiently well performed by the squinting eye, on exclusion of the sound one, to make this method trustworthy.

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[1] Should the reader desire to pursue the theories of amblyopia farther than they are here discussed, he is referred to the papers of Hansen Grut, Schweigger, Schmeichler, and others.

Double Images.—By determining the relation of the image of the squinting eye to that of the fixing eye, the diagnosis of the actual position of the squinting eye and the degree of variance of its visual axis from parallelism can be positively made. In amblyopics, the surgeon experiences the greatest difficulty in forcing the patient's recognition of the image of the amblyopic eye because, as has already been stated, he never complains of diplopia, nor is he ever conscious of the false image of any object. Therefore, examination by means of double images would seem to be impracticable, but in our experience persistent efforts have never failed to accomplish the end sought. At first, the patient absolutely refuses to acknowledge the false image, but with the aid of strong prisms, base out, before the squinting eye bringing the false image nearer to the true and by means of colored glasses (particularly those that render the true light dull and indistinct) perseverance will finally be rewarded with the acknowledgment that the object has its shadow. Having once secured this recognition, the subsequent steps present no difficulty. The prisms, bases out (and bases down if necessary), before the squinting eye which will fuse the images will be the prismatic measure of the deviation. By pursuing this course another important factor in the diagnosis, viz., the differences of elevation of the true and false light, is readily and accurately determined.[2] In monocular squint the false image will be on the side of the squinting eye (homonymous) and often below that of the true eye. The lateral deviation equals 20° to 40° or more, and the vertical difference is usually overcome by a prism of 3° or 4°. This determination of hypertropia is necessary to the treatment, even though the result of the operation can only be cosmetic. Clinically, we have found that the vertical squint is not transferred with the lateral in cases of high amblyopia, the eyes retaining the same relative position even when the sound eye is covered.

Therapy.—The management of monocular esotropia is medicinal in the developmental, and operative in the confirmed stages.

The medicinal treatment is applied with the hope of accomplishing two purposes, namely: the subsidence of the squint, and the cure of the amblyopia. Early in the history of the squint the peripheral cause, hyperopia, can be set aside by paralyzing the accommodation, thereby silencing the abnormal stimulus to convergence always consequent upon the unconscious but unceasing overaction of the hyperopic ciliary muscle. The drug usually employed for this purpose, and the one that is best borne by children without serious results after prolonged application, is atropin, which, it must be said, is not without its disadvantages. The pupils partake in the paralysis and become widely dilated, giving rise to photophobia; the follicular glands of the conjunctiva take on an hypertrophic inflammation; and the squint, for a time at least, seems to be creased because, although the impulse to accommodation not interrupted, accommodation itself (response to the npulse) is impossible. On the other hand, the associated npulse to convergence is greater because of the patient's forts to correct his defective vision by calling urgently n his ciliary muscles. However, this effect of atropin is ansient and may with safety be disregarded. It may be oplied either to both eyes or only to the fixing eye. In e latter case inducement is given the patient to use the uinting eye, provided that its vision is better than that the fixing eye when atropinized. For the same reason, eriodic wearing of a bandage or screen over the sound eye advocated to promote the retinal functions of the deviing eye. It is our opinion that in monocular squint with s attendant amblyopia, this preliminary treatment, while ot often effectual in curing the deformity or in restoring nocular vision, is certainly useful in preventing an increase in the ametropia and a decrease in the vision of e amblyopic eye.

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[2] Cases that prove exceptions to this rule are those of false projection (the so-called second fovea), and those of mental incapacity.

The therapy of esotropia further includes the optical orrection of the refraction error, even in very young ıbjects.[3] A full correction, or as nearly full as possible etermined with the retinoscope and ophthalmometer in ttle children), should be worn *constantly*. The beneficial fect of glasses may be increased by the joint use of ropin. And yet, however essential these means may e in the early treatment of monocular esotropia, they e seldom final, either singly or jointly. As long as the sion of the fixing eye is more acute than that of the uinting eye, the patient will prefer to use the better rgan, and neither the squint nor the amblyopia will be ured; although much progress in this direction will often be secured by employment of the stereoscope, as advocated by Landolt, for education of the fusion faculty. Failing i this, surgical interference becomes justifiable. At thi point several questions are suggested, viz.: At what age i operation to be recommended? To what muscles shoul the treatment be directed? Should it include one or bot eyes?

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[3] Gould has recently (*Phila. Med. Jour.*, May 18, 1898) reported cases in which asses were successfully worn for squint as early as the 29th month of life.

Authorities state that six years is the earliest age a which operation ought to be performed, and that nine to te is the most advantageous. The advocates of early operation urge that a restoration to parallelism of the visual axe will prevent the development of amblyopia, which the claim ensues from non-use. The objections to early operation are: the immaturity of the globes, the natural increase in the power of the muscles of growing children the impracticability of estimating the degree of the squin or of the amblyopia, and hence the effect desired or secure by operation; also the fact that when tenotomy of the internal recti is performed crudely, too extensively, or at to early an age, divergence often ensues between the twentiet and thirtieth year. In esotropia, as in esophoria, the surgeon has the choice of tenotomy of the interni, or advancement of the externi, or a combination of both. Inasmuc as a scientific restoration of binocular vision cannot be obtained in monocular squint, and as all procedures are carried out with the object of removing the deformity, it ha been argued that attention should be directed mainly t the squinting eye. But tenotomy of the internus of thi eye only will be productive of little good, and it will nearl always be necessary to tenotomize the internus of th sound eye also. While in many instances the visual axe may be brought to parallelism by these two operations, i will often be accomplished only by such thorough divisio and separation of the interni from the neighboring tissue hat the effect is practically that of parálysis. In a few ears the evil consequences of such measures crop out ither in divergence of the amblyopic eye, or serious imairment of the inward rotation of both globes. The proeeding that appears to offer the greatest advantages, both mmediate and permanent, is a moderate tenotomy of each nternus (confining the section to the tendon insertion and voiding the radiating fibers and capsular attachments), ombined with advancement of the externus of the deviting eye. If vertical deviation coexist, as it nearly always oes, it is good practice to divide the superior rectus of the quinting eye first, since it has been shown in many cases hat when hypertropia has been eliminated esotropia diminshes, and sometimes even disappears.

ALTERNATING ESOTROPIA.

Synonyms.—Concomitant internal squint or strabismus, lternating internal squint or strabismus, and insufficiency f the externi. (It is to be hoped that the expressions "conomitant," and "strabismus," as applied to esotropia, will oon be discarded.)

Alternating esotropia differs from the monocular form n its pathology, its clinical aspects, and its treatment. For nstance, disease of the foveal fibers of the retina and optic erve of one eye (the result of congenital causes, of accients during delivery, possibly of non-use of the eyes) by ome authorities given much prominence in the developnent of the monocular, play no part whatever in the alterating form; in the latter variety, therefore, amblyopia in its strict sense) is not to be expected. Moreover, in lternating esotropia the squint is perfectly and readily ransferred from one to the other eye. Finally, the results f treatment, both in permanently correcting the deviation nd in reestablishing binocular vision, are infinitely more avorable in this variety.

As to etiology, it may be said that an inherited deficiency of coördination in the cortical centers of fusion is undoubtedly a factor contributing to the loss of binocular fixation, although the usual exciting cause is an hyperopia of from 2.00 to 4.00 D. Inasmuch as all persons with hyperopia do not squint, it has been claimed that there must be other causes; however, close examination of the muscular conditions in hyperopes who do not squint will show that a want of equilibrium is extremely common, and that, while it does not necessarily amount to an actual turning, an inward tendency of the visual axes is unmistakable. Exceptionally, the degree of hyperopia is much higher than 4.00 D., but esotropia is uncommon in very high hyperopia, unless the latter is attended with congenital disease of both foveæ. (Reber.) As in esophoria and monocular esotropia, the overaction of the ciliary muscle necessary to acute vision stimulates the nucleoli of the 3d nerve, and through them the muscles governed by that nerve, with a resultant inward and upward deviation of either eye indifferently; therefore, the squint is transferable.

Diagnosis.—The methods employed in the recognition of monocular are equally applicable to binocular esotropia, and their detailed description may, therefore, be omitted.

As has been said above, fixation is quite indifferent, and the slightest circumstance may suffice to so disturb it that the squint is taken up by the eye which, up to that moment, had been fixing. It cannot be too strongly emphasized that the upward squint is also transferred as can be readily demonstrated by testing with glasses of different colors. For instance, with a red glass before the right, and a blue glass before the left eye, recognition of double images will be almost immediate. If the right be the fixing eye, the blue light (L. eye) will be to the left side of and *below* the red light. However, if the patient's attention is now directed to the blue light, making it the fixing eye, the red light (R. eye) will be to the right of and *below* the blue one, clearly showing that each eye deviated not only *in* but also *up* when the other was fixing. The lateral deviation varies from 20° to 30° and sometimes even 50°, while the vertical deviation is usually corrected by a prism of from 3° to 4°. In conducting this test the surgeon will have but little difficulty in bringing the patient to recognize the two images if vision is anywhere near equal in the two eyes, and after diplopia has been induced, no method of diagnosis is more time-saving or accurate. In children of suitable age for operation the false light will occasionally be suppressed at first, but after some little effort it will be recognized.

It will thus be seen that a description of esotropia naturally includes a description of hyperesotropia, and that internal squint as a pure lateral deviation rarely exists as a functional anomaly. The upward deviation has been noticed by few and overlooked by many writers, for the reason that the inward turn gives rise to a deformity so great that it masks the comparatively insignificant upward turning.

Treatment.—It is not uncommon to perfectly and permanently cure esotropia in small children by paralyzing the accommodation for some months[4] early in the history of the squint; it is scarcely necessary to add that the patient should wear, from the earliest possible moment, an accurate correction of his hyperopia or hyperopic astigmatism.[5] The act of binocular vision is favored by the normal bilateral use of the accommodation which the glasses re-establish, and by the increased visual acuity which they confer.

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[4] Atropin, duboisin, scopolamin, or any other cycloplegic may be used, according to the caprice of the surgeon.

[5] In esotropia complicated with emmetropia or myopia, operation alone offers hope.

These means should never be omitted, even in those in whom the squint seems to be fixed. Failing in these means, resort must be had to

Operation.—Here again, we have the choice of several procedures, namely, tenotomy of one or both interni; advancement of one or both externi, or combined tenotomy and advancement. If hypermetropia underlies esotropia, it would seem rational and logical to employ measures that will reduce the power of adduction, seeing that abduction is not inherently but only relatively weak. We, therefore, give the preference to tenotomy; the more so, as our experience with resection or advancement alone has not led us to look with favor upon it in bilateral esotropia. Moreover, we prefer that each internus should be divided at the same sitting, and that the patient should not be rendered unconscious by a general anesthetic, but should be tested as often as is indicated during the operation, to determine the effect of each snip of the scissors by the change in relative position of the false and true images. If careful double tenotomy does not restore binocular fixation, advancement of both externi is in order, so performed that the power of each externus shall be increased in equal degree. By thus directing any necessary surgery to both eyes, their subsequent relative rotations remain unchanged, and each muscle will respond equally to nerve-stimulation.

The outlook is not unfavorable. Preliminary paralysis of the accommodation and wearing of a full correction of all hyperopia and astigmatism, followed by bilateral tenotomy or advancement, if necessary, will frequently result not only in removing the deformity, but in the reestablishment of what is of vastly more importance and benefit to the patient—the function of binocular vision.

Section II.—EXOTROPIA.

Exotropia may be

1. *Organic*, as a result of destruction of the nucleus or trunk of the 3d nerve either from traumatism or disease; also as a result of direct traumatism to one of the internal recti.

2. *Partly organic and partly functional*, as in partial loss of vision in one eye from anisometropia or antimetropia, or from injury to the eye; in excessive myopia from elongated eyeballs; in those exotropias the result of too free tenotomies for esotropia in childhood; and in high-grade orbital asymmetry.

3. *Purely functional*, due to the influence on a congenitally weak fusion force (desire for binocular vision) either of some refraction anomaly (usually myopia) or of the natural divergence of the orbital axes in adult life.

Classes 1 and 2 may be binocular, although the deviation is usually confined to one eye.

Class 3, the truly functional exotropias, are almost always binocular, and may be defined as a partial loss of the power of convergence, although the vision of both eyes is alike or nearly alike.

The organic exotropias (class 1) have been disposed of in a previous chapter; also such of class 2 as are mainly organic in type. Only those of the second class, depending upon the elongated eyeballs of excessive myopia, or upon asymmetrical orbits, will be mentioned here, and but briefly, the main question under consideration being true functional exotropia.

As has been shown in the previous chapter, esotropia is an active phenomenon growing directly out of overaction of the muscles under the domination of the 3d nerve.

Exotropia, on the other hand, while due to an anomalous condition of the same group of muscles, is a passive phenomenon, and may in general terms be regarded as a *relaxation* of all the muscles governed by the 3d nerve, giving rise to divergence of the visual axes.[6]

It commonly affects both eyes in the earlier stages, in which case it is known as bilateral, binocular or alternating exotropia. In its final phases it is usually confined to one eye, when it is known as monolateral or monocular exotropia.

While the etiology of the affection sometimes seems obscure, the fact seems to stand out that its origin is more or less closely bound up with the presence or development of myopia, which, because of the increased length of the eyeballs and by lessening the demands on accommodation (and hence action on the part of the ciliary branch of the 3d nerve) lessens in like degree the action of the remaining muscles supplied by the 3d nerve, and the eyes tend in the direction given them by the normally innervated external rectus and superior oblique muscles, namely out and down. Thus a deviation of the visual axes not only *out* but also *below* the horizontal plane is likely to be encountered in this, the myopic variety of exotropia.

Should the anomaly be but a functional one up to the time of puberty or soon thereafter, the *deviation tendency* is likely to then become a *true deviation* because of the natural divergence of the orbital axes, which, as has been shown by Weiss, is the feature in the growth of the adolescent and adult orbit. To this latter fact, and to occasional overaction on the part of the superior oblique and external rectus muscles, must be ascribed the exotropia found in the presence of emmetropia and hypermetropia.

[6] The condition is also known as external squint, divergent squint, strabismus divergens, concomitant or alternating external squint or strabismus, and insufficiency of the interni. It is to be hoped that these synonyms will soon give way to the nomenclature here adopted.

It will then be readily seen why exotropia is rarely met with in children (save in high congenital myopes) and why it commonly reveals itself during adolescence or early adult life. Too, the slow departure of the accommodation in the youthful myope, and in other cases the very gradual increase in divergence of the orbital axes, give to the approach of exotropia an insidious form, while esotropia on the other hand is relatively rapid of development. Furthermore, esotropia in the child often becomes esophoria in the youth and may pass through the various degrees of esophoria into muscle-balance when maturity is reached; whereas exotropia in the young or adolescent can only become accentuated as the years wear on. The natural tendency of myopic eyes toward divergence is caused in part by the elongated globes, which must add to the weakness of the adducting muscles in all near work, and in part by the absence of the impulse to accommodation to aid convergence. Very often there is marked irregularity or inequality in vision, and in some cases one eye is so nearly blind that it wanders outward simply in obedience to the tendency impressed upon it by the outward direction of the axis of the orbit. In any case there must coexist a lack of power of the adductors to render the deformity possible, while refractive conditions play a rôle which has been sufficiently explained.

Symptoms.—The only noteworthy symptoms of exotropia are the deviation, the occasional amblyopia, and the defective vision consequent upon the myopia which usually completes the picture. The deviation itself produces no distress and is often borne for years without any special inconvenience to the patient. Rarely is diplopia present, and then mainly when exophoria is passing over into exotropia. Even under such circumstances the patient is only occasionally conscious of diplopia, and while some patients may be educated into seeing double images, in most instances it is difficult to excite them.

Amblyopia, in the sense of the amblyopia of esotropia, is only rarely a symptom of outward deviation, and if it does occur it is a question as to whether it is of the same origin as the esotropic variety. It has been argued that it grows out of suppression of the image of the deviating eye, habitually resorted to by the patient to avoid the confusion of diplopia, and that if suppression of the images falling upon the retina of the unused eye can be so readily learned as it is by microscopists, ophthalmoscopists, astronomers and others, it seems reasonable to assume that it is resorted to, unconsciously but none the less surely, by exotropics (as also by the victims of longstanding ocular palsies or pareses) to avert the confusion of diplopia. We have no means of learning whether the esotropic infant or child has ever seen well with its squinting eye before that eye deviated; nor whether the complexity of functions that issue in binocular vision have ever been thoroughly acquired and brought into harmonious automatic relations. Exotropia, on the other hand, seldom reveals itself before puberty—long before which time binocular vision has become one of the cardinal functions of the central nervous system. Let it be remembered that the performance of binocular vision calls for harmonious synchronous action on the part of areas in the motor region of the cortex, also on the part of a large portion of the occipital cortex, and lastly on the part of the 2d, 3d, 4th, and 6th (namely four out of twelve cranial nerves. With this proposition in mind, it is not difficult to conceive that many esotropic children have never thoroughly acquired this complex function, and that in such children a moderate degree of hypermetropia suffices to so disturb the act that the cerebral end of the apparatus finally abandons the struggle. Hence we have spoken of a congenitally weak fusion-force. Similarly in exotropia, at least the myopic variety, when the impulse to accommodation is no longer excited, another disturbing factor has been engrafted on the act of single vision and the outward deviation of the eye or eyes is the signal of the surrender of the higher centers. However, in exotropia we are dealing with eyes that have enjoyed a high degree of visual acuity, and deviation certainly means diplopia with its sequels unless one eye learns to disregard its images. Necessarily this has naught to do with the amblyopia of those outward deviations which are the result of overdone tenotomies for esotropia in childhood, and which are to be regarded more as pareses of the operated interni than as true exotropias.

It is proper to state that in exotropia there may be ocular symptoms, but these commonly result from the refractive error present, and generally disappear after its correction.

Diagnosis.—The diagnosis of exotropia rests upon inspection, the cover-test with and without prisms, and the study of the double images (when present) with and without prisms. Simple inspection will in most cases not only determine the presence of the anomaly, but may also show which is the fixing eye and what the probable degree of deviation. Resort, however, is generally had to the cover-test as follows: The patient is seated facing a candle-flame or some similar small bright object 20 or more feet distant. A small piece of card-board is then carried from one eye to the other, and if each eye fixes as its fellow is covered, the squint is of the alternating variety. When both eyes are uncovered it will be found that the patient consciously or unconsciously uses either the right or left eye by preference for fixation. We therefore speak of it as the fixing eye. The fellow organ then assumes a varying degree of divergence. Should the squint be of the monocular or monolateral variety, it will be found, on applying the cover-test, that the deviating or amblyopic eye makes little, if any, movement when the fixing eye is covered. Alternating exotropia may be measured with fair accuracy by using prisms in connection with the cover-test. Having the patient fix on the candle-flame or other object 20 feet distant, a 10° prism, base *in*, is brought before either eye, preferably the fixing one. Applying the cover-test, it will be found that the excursion of each eye as the cover is shifted from side to side is much diminished, and by increasing gradually the prism strength, base in, a degree will finally be reached at which both eyes are apparently steady as the cover moves back and forth. Not infrequently it will be found that a vertical deviation coexists, when it will be necessary to learn the prism needed to stop the vertical as well as the lateral movement. The prism or prisms required to stop all movement of the eyes under cover, will be the prism measurement of the squint; and if, in carrying out the test, very high degree prisms are needed, the effect can be divided between the eyes. Those not familiar with the test will do well to guard against over-correcting with prisms, as this will induce esotropic movement under the cover and thus lead the surgeon into serious error.[7] The test is purely objective and of great service.

When double images can be excited, we have another valuable test at hand. By coloring the image of the fixing eye with a cobalt glass, the relative position of the images is easily studied, and the degree of the prism that will bring the images into a horizontal or vertical line (or both readily learned. However, this measure is applicable only to those patients in whom double images can be induced, and who can coöperate mentally with the surgeon in the study of the case; whereas, the cover-test, combined with prisms, renders the surgeon absolutely independent of the patient, and is of signal service in all cases of alternating exotropia irrespective of age or mental condition.

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[7] The same mistake may be just as easily made in estimating the prism degree of esotropia or of right or left hypertropia.

Treatment.—The management of exotropia is optical and operative. At first thought it would seem that treatment might be also therapeutic; that, if atropin serves to interrupt the pathologic process in the early stages of esotropia, pilocarpin or eserin ought, logically, to be of service in the same period in exotropia. Theoretically, this reasoning is correct, but unfortunately it is not borne out by clinical facts. In esotropia, we are dealing with a force that needs curbing, and atropin is the ideal agent. In exotropia, we have to do with functions (accommodation and convergence) that are waning, or with conditions that are the result of anatomic changes (divergence of the orbital axes, and elongation of the globes). The fact is that myotics by stimulating accommodation alone would rather lessen the associated stimulus to convergence, and thus add to the divergence of the eyes. Moreover, it would seem that the instillation of a very weak mydriatic might, by inducing extra stimuli to the ciliary muscle, incite the adductors to action, and hence be of service in the early stages of exotropia, although there is no record of such treatment directed to early exotropia.

The optical treatment of exotropia may be effected by correcting lenses alone, or in combination with prisms. The greatest care should be observed to learn the exact static refraction[8] in every case, when the following rules are in force:

For non-presbyopes. All myopes under 6 D. to be given a full correction of their optical defect, that the accommodation may be forced into action. It will often be of advantage to assist the accommodation at first with one drop of a one grain to the ounce solution of pilocarpin muriate in each eye three times a day.

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[8] Refraction under complete cycloplegia.

Myopes of over 6 D. to be given the fullest possible correction consistent with any degree of comfort in work at the near point. Pilocarpin is also of service in enabling these patients to become accustomed to the new conditions imposed.[9]

Hypermetropes of whatsoever degree to be given the weakest spherical correction consistent with comfort in working at the near point.

Presbyopes to be given the strongest minus spherical (if myopes), or the weakest plus spherical (if hyperopes) that will enable the patient to conduct work at 13 inches, with pilocarpin (or eserin) again as a synergist.

The object of all the foregoing measures is to induce action of the ciliary muscle in the hope of arousing an associated stimulus to the adductors, and thus lessening the divergence.[10] If eight to twelve weeks' constant wear of the correcting lenses does not dissipate the exotropia, training of the convergence after the method advised in the chapter on exophoria will be in order, but this is feasible only in the lowest grades of exotropia where binocular vision is still possible. Anything over 15° falls out of this class. As a last optical resort, stereoscopic training of the adductors may be tried after the method of Landolt, and some few patients will respond to this method of educating the fusion-impulse after all other methods have failed. Finally, if, after optical correction, the deviation be less than 15°, and does not respond to convergence training, or if it is over 15°, operation is the final resource. Inasmuch as we are dealing with a passive phenomenon, shortening of the relaxed muscles is indicated, rather than tenotomy of their antagonists, hence advancement is preferred to tenotomy. In monolateral exotropia, the deviating eye should always be approached first. If vertical deviation coexist, it should be remedied before proceeding to advancement of the internus of the squinting eye, and if these two steps leave a residual deviation, tenotomy of the externus of the squinting eye may be performed. Any deviation then remaining must be met by advancement of the internus of the sound eye. Rarely will it be necessary to go on to tenotomy of the externus of this eye.

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[9] In a 20-year-old young woman who came under our care, wearing — 10.00 for distance and — 6.00 for near work, the patient was relieved entirely of her asthenopia and incipient exotropia by two months' use of — 13.00 D. S. ⌒ — 1.00 Cy 180 in each eye (her full correction), the employment of pilocarpin as above, and training of the convergence with prisms.

[10] It should be mentioned that this adaptation of the correcting lenses to the exotropia applies to their spherical components only. Any cylindrical element necessary should be prescribed in its full static strength.

In binocular exotropia, the muscles to be attached are taken up in the following order: Any vertical imbalance must be first corrected; efforts to restore the lateral balance are then made by advancement of the internus of the eye which does not fix; or, if fixation be indifferent, by advancement of the internus of that eye which presents the poorer corrected vision. If this prove insufficient, advancement of the internus of the fellow-eye becomes necessary, and if external deviation persists despite all these procedures, tenotomy of either or even both externi is in order. These latter measures, however, will be required only in the most aggravated exotropias.

As a rule, exotropes who have once possessed the faculty of binocular vision are much more easily restored to its privileges than young esotropes who, in all probability, have never enjoyed good binocular vision. Hence, the prognosis of the operative treatment of exotropia is favorable, although the length of time necessary to bring the visual axes into coördination extends in some cases over several months.

Section III.—HYPERTROPIA.

Hypertropia, or true vertical deviation of one visual axis above the other, is not often encountered alone, but is usually associated with either esophoria or exophoria. Admitting the relative infrequency of pure uncomplicated hypertropia, one cannot but be struck with the meagerness of references to this anomaly in the most recent as well as in the older text-books on ophthalmology; nor does recent ophthalmic literature throw much light on the subject. And the foregoing fact is even the more striking when it is remembered that the same causes which underlie the *functional* or latent vertical imbalances (hyper*phorias*) lead directly up to, and are as distinctly causative of, the actual vertical deviations or hypertropias. For instance, the ciliary overaction which in hypermetropic eyes often gives rise to a temporary esophoria or hyperphoria, may, under certain conditions, carry the anomaly from the latent or heterophoric class over into the actual or heterotropic phase; in other words, long perverted physiology finally issues in pathology. Just so long as the patient's fusion-power asserts itself, just that long will the patient preserve binocular vision and, in all likelihood, present a train of ocular or reflex symptoms as the result of the extra output of nerve-force necessary to maintain fusion. During this stage *deviation tendencies* only can be elicited—in other words, we are dealing with heterophoria; and many patients will carry a defect of this kind throughout a life of large usefulness. But it frequently happens in childhood or adolescence that the supply of nerve-force necessary to fusion is not equal to the demands, and the muscular system of one or the other eye gives up the unequal struggle and latent tendencies become manifest defects, or heterophoria (functional turning) advances now to heterotropia, actual turning or squint. This variety of squint results in the vast majority of cases either from uncorrected errors of refraction or from the change in the dimensions and direction of the orbital axes common to the time of puberty. Hence, hypertropia is similar in origin to esotropia and in many respects to exotropia, with either one of which it is therefore nearly always associated.

In hypertropia either the right or the left visual line may be habitually higher than the other —right and left constant hypertropia respectively; or each visual line may be alternately higher than the other—alternating hypertropia. Vertical squint is further classified as strabismus sursumvergens when the lower eye is the one that habitually fixes, and strabismus deorsumvergens when the upper is the fixing eye. The preceding statements made in particular reference to the etiology of regular hypertropia (a deviation of one visual axis above the other) seem to us none the less applicable to a certain remarkable class of cases, mentioned by Stevens,[11] in which *both* visual axes deviate either above or below the horizontal plane when the head and eyes are in the primary position. Deviation of both axes upward is termed anatropia. That is, in anatropia, the right eye deviates up behind the screen or cover when the left is fixing, the left eye moving up also (instead of down as in hypertropia) the instant the cover is carried to that eye and the right eye is permitted to fix. Katatropia is present when with the same cover-test the excursion is down in both eyes.

It is our belief that anatropia is an overaction phenomenon and that in all probability it is closely related to esotropia, a similar phenomenon. The excessive impulse which in hypermetropia is sent to the ciliary muscle, affects in like manner the remaining muscles supplied by the third nerve, in consequence of which the eyes tend in the direction resulting from the combined action of those muscles, namely, up and in. If in esotropia the cover test be used with the patient fixing on some point 20 or more feet distant, it will be found that both eyes deviate not only inward but also upward as the cover is shifted from one to the other eye. On the other hand, the absence of impulse to the ciliary muscle of myopes (who commonly use little if any accommodation) carries with it a very much diminished impulse to the other muscles governed by the 3d nerve, and their resultant inaction permits the eyes to be deviated in the direction resulting from the combined action of the remaining ocular muscles (the external rectus and superior oblique), namely, down and out. The cover-test in such a case, provided binocular vision can be maintained, will show that both eyes deviate both outward and downward under the cover or screen as it is carried from one to the other eye.

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[11] *Annales d'Oculistique*, April, 1895.

The organic hypertropias, generally paralytic, are seen principally in grave cerebrospinal disorders and have been considered at some length in the chapter devoted to palsies. These forms of hypertropia must not be permitted to obscure our ideas about true right and left hypertropia and strabismus sursumvergens and deorsumvergens, whose origin is closely bound up with anomalies of refraction and with the shape and development of the orbits.

Symptomatology.—The symptomatology of the earliest stages of hypertropia is largely that of hyperphoria. However, when the weak depressors or elevators have abandoned their fruitless task, the eye to which they belong will deviate in the direction of action of the stronger muscles, binocular vision will be lost and along with it will most likely disappear much of the patient's ocular and reflex discomfort. The resulting deviation which is the main objective symptom, is easy of recognition in most cases, especially with the cover test at 20 feet. The cardinal subjective symptom is the amblyopia of the squinting eye. In recent or low grade vertical squints, especially if they are alternating, even corrected vision will be compromised to some extent in both eyes, although more decidedly so in the squinting eye. In old cases the fixing eye will nearly always present a close approach to normal vision (corrected if necessary) while the vision of the squinting eye is often lowered to a remarkable degree.

The mooted question as to whether the amblyopia produces the deviation or the deviation the amblyopia is discussed under the chapters on esotropia and exotropia. Aside from the deviation and the amblyopia, there is no symptom worthy of note unless it be the peculiar "wall-eyed" appearance of such patients, especially if the squint be alternating, giving to the patient the well-known hyperphoric or hypertropic "stare." Partial ptosis not ascribable to other cause is regarded by some authorities as symptomatic of hypertropia.

Diagnosis. The presence of this anomaly is easily detected, and in most cases the character of the squint and the eye to which it belongs can be determined almost at a glance. In the alternating variety resort must frequently be had to the cover test to learn which eye fixes by preference. This test will be productive of the best results if carried out in a half-darkened room, using a small gas-flame or some other luminous point as the fixation object. By interposing a weak prism before the squinting eye with the apex of the prism placed in the direction in which the eye squints, and increasing it in strength until the eyes no longer move visibly when the cover is carried from one to the other eye, the degree of the squint may be measured with a reasonable degree of accuracy. This method is applicable also to those cases in which the patient cannot be made conscious of diplopia, and it enjoys the additional advantage of being an objective method, thus rendering the surgeon entirely independent of the patient's testimony. In cases that are or can be made conscious of diplopia the use of the cobalt glass before the sound eye is of great value, and all that will be necessary is to find a prism that brings the two images to a horizontal level. The Maddox rod is employed by some surgeons in the diagnosis of hypertropia, but its use is largely restricted to the estimation of deviation tendencies rather than actual deviations.

Treatment.—The treatment of the varieties of hypertropia under consideration is orthopedic and operative.

By orthopedic treatment is meant the persistent use of a full cycloplegic correction of any ametropia that may be present. The effect of such lenses will frequently be much enhanced by continuing the effect of the atropin or other cyclopegic for from 8 to 12 weeks, in the hope of thus reestablishing proper relations between accommodation and convergence.

In young subjects (under 10 or 12 years) these measures will frequently lessen the deviation, and sometimes dissipate it altogether. Oftener, however, and in subjects over 12 years of age in particular, a considerable deviation remains, which must be met by some operative procedure. The laws governing operative interference in heterophoria are here in force. Overaction hypertropias, mainly those found with esotropia (including anatropia), demand tenotomy of the overacting muscle or muscles of the squinting eye to be combined in some cases with tenotomy of the associated muscle or muscles of the sound eye. Underaction hypertropias (usually hyper-exotropias or exceptionally katatropias) will be best met by advancement or muscle-shortening of the faulty muscle or muscles of the squinting eye, and, where this is insufficient, by a similar procedure on the muscle at fault in the fellow-eye. It is to be remembered that division of the superior rectus acts by association on the levator palpebræ superioris, and is followed, not only by depression of the cornea, but also by lifting of the upper lid, in consequence of which a considerable amount of sclera will be exposed above the cornea. However, this fact may be utilized in cases of partial ptosis, both to aid the elevator of the lid as well as the depressor of the cornea. It is further to be borne in mind that operations on hypertropes over 12 years of age are frequently cosmetic operations pure and simple; and, notwithstanding Stevens's claim that restoration of binocular vision should be the ultimate aim in such cases, such termination of them is rare and must be viewed only as a most fortunate circumstance.

Finally, operative treatment is contraindicated in hypertropia of variable degree; or of recent origin and progressive; in paretic cases of central nervous origin; and in rheumatic or gouty individuals well on towards 50 years of age.

PART IV.

OPERATIONS ON THE MUSCLES.

CHAPTER I.

TENOTOMY.

THE object of tenotomy is two-fold: By altering the endinous attachment of a muscle to change its mechani- al relations to the globe and to the other muscles, and by hus lessening the power of the muscle to so influence the listal response to innervation, that equilibrium and co- rdination shall be inaugurated or re-established; second, o develop or restore symmetric and corresponding nerve- xcitation. We are concerned, first, with the muscle or nuscles at fault, and, second, with the degree of deviation, . *e.*, whether there shall be partial or complete muscle- livision, and whether it shall apply to one or both eyes. The solution of these questions becomes comparatively imple when, without reference to prisms or other re- racting media, the patient will recognize double images, ne of which falls on the fovea of that eye, the visual line f which is directed toward the object that is focused the true image), the other image falling on a part of the etina of the deviating eye that is removed from the foveal egion (the false image). As the oblique muscles are arely divided, and the lateral oftener than the vertical nes, a description of tenotomy of the internus will suf- ice for all. Unless there is good ground for believing hat one muscle is abnormal, either in structure or inser- ion, the operation should in all cases be divided between he two muscles, relieving an equal amount of tension in ach. The evils resulting from extensive tenotomy on ne eye for high-grade convergent strabismus are limited novement nasalwards, diplopia in the periphery of the field, cicatrization of the conjunctiva and capsule at the side of the wound, retraction of the caruncle, and protrusion of the ball.

Anesthesia.—In heterophoria, when the success of the operation depends on a partial or accurately measured tenotomy, local anesthesia is essential. Indeed, a precise operation cannot be performed unless the patient is conscious and can advise during the procedure of the effect obtained by the various steps. Between cocain and holocain we have no choice, although our limited experience with the latter would lead us to believe that the final stages are less painful with holocain when the field of operation is bloody and deep tissues are to be incised. In this connection must be mentioned also the infusion of extract of suprarenal capsules, which, by reason of its powerful vaso-constrictor properties, is not only of admirable service in forestalling any hemorrhage that might obstruct the field of operation, but is said by Terson to possess faint anesthetic qualities itself. It is well borne by the eye. Eucain we have never resorted to in tenotomy, because of the smarting its instillation causes, and because hemorrhage would probably be much more profuse than with either of the other two local anesthetics. In heterotropia, where greater effect and less accuracy is desired, and particularly in children, general anesthesia may be substituted.

Antisepsis and Cleanliness.—The operator's hands and nails should be scrubbed with warm water and soap until all mechanical impediments to the action of the chemical agent to be used immediately afterward are completely removed. The hands are then dipped in a solution of bichlorid, biniodid, or cyanid of mercury, 1 to 3000. The skin of the patient's face in the neighborhood of the eye is cleaned with warm water and soap, dried with a sterilized towel or piece of gauze, and washed finally with the mercurial solution. In this manipulation, particular attention must be paid to thorough cleansing of the eyebrows and lashes. The conjunctival sac and the conjunctival surfaces of the lids are bathed in a physiologic salt or saturated boric-acid solution, and fresh cocain solution is applied. The instruments are first placed in boiling water and then in alcohol, where they are allowed to remain until used. This simple method of treating the instruments is sufficiently germicidal for all operations where the field is not unusually septic. After insertion of the speculum or separation of the lids by an assistant (lid elevator held in one hand and the lower lid depressed by a finger of the other hand) the conjunctiva and the capsule lying immediately over the insertion of the tendon into the sclera are firmly grasped by the single tooth conjunctival forceps. Considerable pressure with the forceps at right angles to the ball is necessary to ensure the embrace of the capsule with the mucous membrane. The advantage of securing both can be appreciated in the next step, the incision. If both structures can be divided and the tendon exposed at its insertion by one snip of the scissors, this part of the operation is considerably simplified. Two slight difficulties present themselves in case the conjunctiva alone has been divided: the smooth surface of the capsule uncovered by mucous membrane and made tense by outward rotation of the cornea, is slippery and hard to grasp; and again, unless one is cautious he is apt to include the tendon in the grasp of the forceps and to divide it unintentionally. The incision should not exceed the width of the tendon; if the desired result is not obtained it may be widened at the close of the operation; if it be too narrow, the necessary manipulations are hindered. A few operators prefer to make the direction of the incision horizontal, parallel with and close to the lower border of the tendon, but the common practice, and the better one, is to make it vertical in order that it may have greater influence in the regulation of the final result. Having exposed the tendon at its attachment, a small hook is passed under its entire width from above or below, the distal end of the hook coming into view at the upper or lower extremity of the incision, using the scissors to snip away any capsular folds that the hook may push before it. The tendon may now be divided to any extent desired (Fig. 24). The method recommended for the correction of heterophoria is to incise first the middle fibers and then the marginal, according to the degree of effect previously determined to be necessary, gauging the progress toward equilibrium by frequent interruptions of the operations for examination by the tests above described. In heterotropia, the division of the operation into these stages is less important since the tendon is to be detached in its entire width. If section of the tendon alone will not restore equilibrium, the conjunctival wound can be enlarged, and the fibers radiating from the upper and lower margins of the tendon and from the capsule of

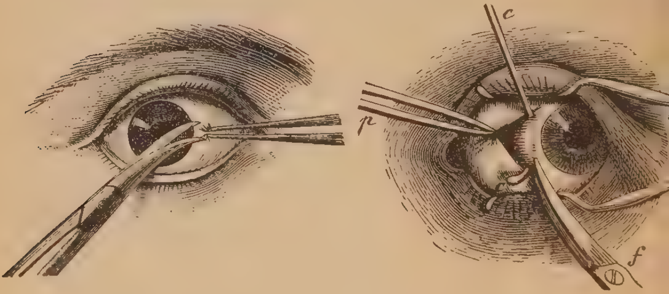

FIG. 24.—After Meyer.

Incision of the conjunctiva. Section of the tendinous insertion.

Tenon can also be severed, but care must be exercised that the evil effects of too extensive division of the capsular attachments already alluded to shall be avoided. The value, in prism degrees, of section of the tendon varies in different cases from 2° to 10° or more. In graduated tenotomy, *i. e.*, division of a few fibers in the center of the tendon, the effect is decidedly limited; when the whole tendon is severed, without disturbing the conjunctiva or capsule, the effect would average about 6°. In proportion to the tissues divided and the extent of the wound the effect may vary from slight weakening of the muscle to a practical paralysis. Union of the tendon to the sclera may take place so far back that the muscle loses all power of rotating the cornea, or, indeed, it may always remain detached. Perhaps our figures seem small, but they are the result of an experience numbering several hundred tenotomies and they are, we believe, as correct as general averages usually are.

The after-treatment is simple. Cold applications for a few hours and frequent instillations of saturated boric-acid solution are all that is necessary in the majority of cases. A bandage is seldom useful; on the contrary, it may be positively harmful in that, in the exclusion of binocular fixation, the important factor to success, namely, the unconscious effort at fusion, is prohibited. If immediately, or a day or two after operation, the defect is found to have been over-corrected, a suture to either close the conjunctival wound or a deeper one that shall include the capsule and the severed muscle may be inserted. It is the practice of some operators to introduce the suture before cutting the muscle, so that an over-defect may be instantly neutralized; but this is, we believe, an unnecessary precaution for a skilful operator.

Accidents.—It occasionally happens, through an anomalous distribution of the large vessels, that one of them is cut or torn, producing immoderate hemorrhage. In this unfortunate event the operation should be suspended and a pressure-bandage applied. A second accident is the unexpected perforation of the sclera by the scissors while cutting the tendon, which must be ascribed to using too sharp-pointed scissors or to directing the ends of probe-pointed ones at right angles to the tendon (instead of on a line with the muscle), with undue force. The operation must be at once discontinued, the conjunctiva sutured over the perforation, and a pressure-bandage applied.

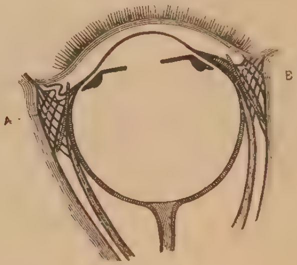

FIG. 25.—*a.* Internal check ligament. *b.* External check ligament.

Healing.—After the usual operation, the tendon re-attaches to the sclera in from three to four days. If the wound should become infected, tenonitis, orbital phlegmon, ulceration of the sclera or cornea, or panophthalmitis may ensue. Tenotomy has been known to be the exciting cause, also, of detachment of the retina, essential phthisis, and hemorrhage fatal to the sight and to the integrity of the ball.

Disappointment as to Result.—A perfectly performed tenotomy will sometimes fail to favorably affect the deviation for which it was undertaken, in which event it is not unlikely that the failure can be ascribed to some anomalous arrangement of the check ligaments which accompany the recti muscles. Fig. 25, from Morris' Human Anatomy, shows very well the connections between the muscles, the capsule of Tenon, and the check ligaments.

CHAPTER II.

ADVANCEMENT OR RESECTION.

NUMEROUS operations for increasing the efficiency o any one muscle have been advocated. Their number point to the inadequacy of any single procedure. The object ma be accomplished either by advancement of a muscle or tendon, or by resection—or cutting out of a piece of a tendo or muscle. Naturally each of these methods has its supporters. Without describing all of them with their various modifications, it seems to us to be enough to describe the on which has proved the most efficient in our hands, namely, modification of Savage's resection operation. Excepting instances of marked deformity, in which the purpose of operation is mainly cosmetic, this measure affords entirely satisfactory results in many cases. Its effect can be accuratel gauged; it is absolutely without danger of increasing th original deformity; although tedious in its performance, it i attended with but moderate traumatism, and the healing is as a rule, uneventful and rapid. The principal objectio to be urged against most advancement operations is, tha the sutures by which the advanced tendon is attached to th conjunctiva frequently tear out, allowing the tendon to recede to a point farther back on the ball than that to whic it was formerly attached, and the original deviation is thu aggravated. The advantage of resection or shortening muscle is that there is no danger of increasing the deformity, since the tendon, at its attachment to the sclera, is no disturbed and the shortening process is confined to th muscle and tendon.

First Step.—After thorough cocainization and the us of extract of suprarenal capsule to prevent hemorrhage, the conjunctiva is grasped with the forceps over the inferior corner of the tendon-insertion and divided vertically 4 to 5 mm., and (starting at the same point) horizontally along the lower border of the muscle as far back as is necessary, to give a roomy field for operation.

The subconjunctival tissue and the capsule of Tenon are similarly treated, the structures thus far divided forming a flap, which is turned upward. (Fig. 26.)

Second Step.—The muscle is now brought plainly into view by the strabismus hook and freed from all adhesions, when a black silk thread, armed at each end with a fine curved needle, is introduced into the substance of the muscle from 6 to 8 mm. back of its insertion, one needle passing through the upper border of the muscle from its conjunctival to its scleral surface, the other through the lower border in like manner, and the thread drawn taut over those fibers included between the two points of entrance of the needles. (Fig. 27, p. 172.)

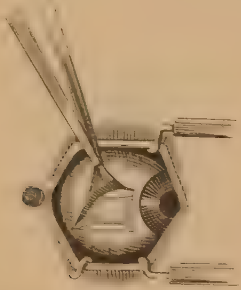

FIG. 26.—Exposure of the muscle.

Third Step.—The needle belonging to the upper portion of the muscle suture is now passed under the tendon at the *upper* border of its insertion and brought out near the center of the conjunctival surface of the attachment; the second needle, belonging to the lower portion of the muscle suture, is introduced in exactly the same manner under the *lower* border of the tendon-insertion and brought out on the conjunctival surface just below the first needle. An assistant now grasps the muscle back of the point where the needles were first introduced and brings the muscle forward, when the suture is drawn tight and tied, by this measure advancing the whole muscle nearly as far as the tendinous attachment and producing a fold or hump in the muscle (Fig. 28, p. 173). The summit of this muscular fold or hump is then cut away, leaving the upper and lower borders intact and the two raw surfaces in juxtaposition. The suture is cut off so that its ends may protrude a short distance through the conjunctival wound, which is now brought together by one or two sutures.

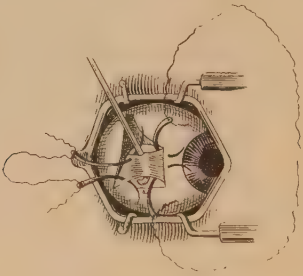

FIG. 27.—Introduction of sutures.

Pain in the healing may be largely prevented by the constant application of ice-water compresses and frequent instillations of a solution of boric acid and cocain. The conjunctival threads may be removed in a few days, and the thread in the muscle should be allowed to remain as long as it causes no irritation. Any difficulty that might be experienced in the removal of this deep thread can be forestalled by leaving long ends to it, so that it may be readily brought away by the forceps.

Capsular Advancement.—Another method of correcting the smaller deviations is to advance the capsule of Tenon, as recommended by de Wecker.[1] It consists essentially in incising the conjunctiva freely in the vertical meridian about 4 to 6 mm. from the cornea; then, grasping the capsule over the insertion of the muscle at fault, sutures are made to take firm hold on the capsule and the free ends are carried under the conjunctiva, one above and the other below, as far as the middle of the cornea. These sutures are then tied with careful attention to the traction they produce on the advanced capsule, so that the tension will be equal above and below. This operation, known as capsular advancement, is indicated only in the smallest degrees of heterophoria, and is of no value in well-established squint.

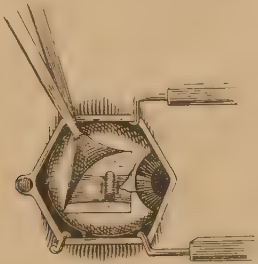

FIG. 28.—Tying of sutures, producing knuckle in muscle.

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[1] De Wecker and Masselon, *Man. d'Ophthal.*, vol. ii, p. 778.

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